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DISEASES OF THE EYE

A HANDBOOK OF OPHTHALMIC PRACTICE FOR STUDENTS AND PRACTITIONERS

BY

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PREFACE TO THE TENTH EDITION

In this, the Tenth Edition of this text-book, the revision includes useful and important observations which have appeared in recent ophthalmic literature, as well as certain statements and recommendations, modified or emphasized, as the case may be, based on personal experience.

Reference to the following subjects is made either for the first time or, having been previously included, has been somewhat elaborated: The Diaphragm Lamp (Gullstrand); Contact Illumination; Binocular Visual Acuteness, and Illumination and Acuteness of Vision; Sunlight as a Source of Illumination in Ophthalmoscopy; Simple Centric Ophthalmoscopy (Gullstrand); Ophthalmoscopy with Red-free Light; Agricultural Conjunctivitis (Patton and Gifford); Striate Clearing of Corneal Opacities; Uveoparotitis; Hernia of the Vitreous; Senile Changes in the Optic Nerve; Temporary Amaurosis in Infants; Butyn; Extraction of Cataract by Suction (Barraquer's method); Subconjunctival Excision of Ptergyium (Ziegler); Tendon Transplantation; Muscle Recession with Scleral Suturing; O'Connor's "Cinch Shortening" Operation.

In certain portions of the book the revision has included a rearrangement, with additions, of the subject matter, for example, in the paragraphs devoted to Iritis, Iridocyclitis, Uveitis, Retinal Angiosclerosis, and the Ocular Muscle Palsies.

The large literature which has accumulated with reference to "slit-lamp investigation" has been utilized, although, naturally, subject to the limitations imposed by a text-book of this character. A certain amount of old matter has been either eliminated or changed in accordance with modern concepts.

A number of the illustrations have been redrawn prior to their reproduction; others have been set aside, and about twenty-five new ones have been inserted.

The author is indebted, as in previous editions, to Lt.-Col. Elliot for the description of Corneoscleral Trephining, and to Dr. William M. Sweet for an account of his method of localizing foreign bodies in the eyeball by means of the *x*-ray. Dr. Edward Jackson has rewritten his Section devoted to Skiascopy, or the Shadow-test.

The author ventures the hope that this revision and these additions and alterations may prove to be satisfactory. Once more he expresses his high appreciation of the reception which the book has received.

G. E. de S.

1705 Walnut Street,
Philadelphia, Pa.
April, 1924.

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DISEASES OF THE EYE

CHAPTER I

GENERAL OPTICAL PRINCIPLES

Transmission of Light.—By light is meant that physical force or form of energy which, acting on the sentient elements of the retina, causes the mental perception of the specific energy, that is, *sight* or *vision.*

From each point of the surface of a luminous body *light* or *rays of light* proceed in straight lines in all directions, and in order to explain the transmission of light it is assumed that throughout the universe there exists an exceedingly tenuous matter to which the term *ether* is applied (see page 21). Exactly what the vibrating disturbances are which constitute light is not certainly known.

Refraction.—By refraction of light is meant the alteration which takes place in the direction of luminous rays, which pass obliquely from one medium into another of different density.

A ray of light passing through air keeps the same direction until it strikes obliquely the surface of a denser medium, when its course is changed toward the perpendicular to that surface. If this denser medium is a piece of glass bounded by parallel sides, the ray, as it passes through the second surface, is bent back again into the rarer medium.

Rays passing from a denser into a rarer medium are deviated from the perpendicular. The ray now has a direction parallel to its original course; the sides being parallel, the deviation at each surface is equal in extent, but opposite in direction (Fig. 1).

If the denser medium is bounded by oblique surfaces, the deviation at the second surface does not restore the ray to its original direction, but it still more increases the alteration of its direction (Fig. 2).

Index of Refraction.—The deviation of the ray from its course depends upon the difference in the density of the two media.

A ray passing obliquely from one medium into another of the *same* density is not bent from its course. The relative resistance of a substance to the passage of light is expressed by its *index of refraction.* The absolute index of refraction is its resistance as compared with vacuum; but as there is very little difference between the indices of refraction of air and of vacuum, air is considered as 1 for all calculations in lenses.

As the difference in the density of the two media increases, the ray is bent more sharply from its course, and the angle it forms with the perpendicular after refraction by a denser medium is proportionally smaller than the angle formed by the ray before refraction.

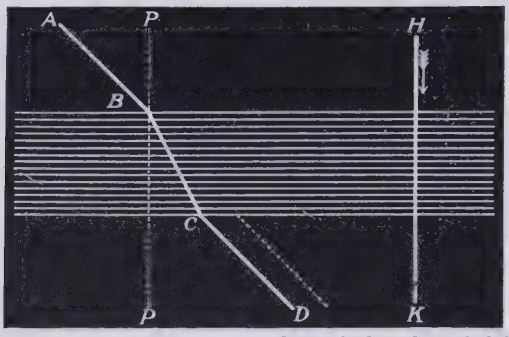


FIG. 1.—Refraction of light through a plate of glass bounded by plane surfaces which are parallel: *A–B* is the incident ray; *B–C*, the same ray, refracted by the first surface, nearer to the perpendicular, *P–P*; *C–D*, the same ray, refracted by the second surface, becomes parallel to *A–B*, its original direction. The ray *H–K*, perpendicular to the surfaces *B* and *C*, undergoes no refraction.

The angle formed by the ray with the perpendicular to the surface of the second medium is called the *angle of incidence*—angle *I*. The angle formed by the ray with the perpendicular after refraction is called the *angle of refraction*—angle *R*. The sine of the angle of incidence, divided by the sine of the angle of refraction, gives the index of refraction. Glass used in the manufacture of spectacles has an index of refraction of about 1.53.

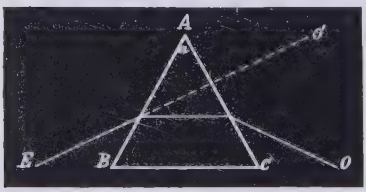


FIG. 2.—Refraction through a denser medium having oblique surfaces. At each surface the ray is bent toward the base of the figure.

Prisms.—A *prism* is a portion of glass or other refracting substance bounded by two plane surfaces which are inclined to each other, forming an angle, which is called the *refracting angle*, or simply the *angle* of the prism (Fig. 2, *a*), and is expressed in degrees. Prisms are often designated by the numbers of degrees in the refracting angle.

The sides of the prism converge to a thin edge at one extremity, called the *apex* (Fig. 2, *A*); at the other extremity they diverge from each other and form the *base* (Fig. 2, *B–C*).

Refraction Through a Prism.—If a ray of light from an object (Fig. 2, *O*) passes through a prism the refractive index of which is greater than air, the deviation is always from the apex toward the base of the prism.

To the eye of an observer placed at the other side of the prism

(Fig. 2, *E*) the refracted ray seems to come from the direction of the apex (Fig. 2, *O'*), since a ray is projected backward over the course given to it by its last refraction, and a single object appears double if, with both eyes open, a prism of sufficient strength is placed before one of them. The angle which the ray in this last direction forms with the ray in its original direction is called the *angle of deviation.*

When one eye, on account of muscular weakness, is unable to direct its visual line to the point of fixation, a prism will alter the direction of the ray from the point of fixation so that it coincides with the visual line of the weaker eye. The refractive properties of a prism are utilized to test the strength of the ocular muscles, that is, to ascertain prism-convergence, prism-divergence, and sursumvergence (see pages 77 and 78), to estimate the balance of the exterior eye muscle (see pages 77, 80, and 639), to neutralize the diplopia caused by abnormal deviation of the visual line—for example, in paralytic strabismus and in the treatment of heterophoria (see page 643)—to detect malingerers who feign monocular blindness (see page 583), and in the application of the Wilbrand-Saenger test to determine the situation of the lesion in hemianopsia (see page 599).

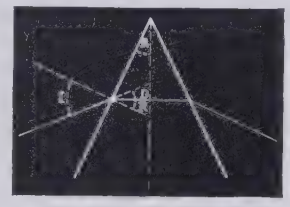


Fig. 3.—Deviation produced by a prism: *I*, Angle of incidence; *R*, angle of refraction; *D*, angle of deviation; $R + D = I$; *D* equals in weak prisms about $\frac{1}{2}$ of *R* (Jackson).

Angle of Deviation.—The angle of deviation is the angle formed by the incident ray with the refracted ray. The amount of this angle is somewhat more than one-half of the refracting angle of the prism for all prisms between 1° and 10°, but for practical purposes the two may be considered equal. Above this the deviation rapidly increases.

When the angle of incidence, formed by a ray in the interior of a prism, amounts to 40° 49′, the angle of refraction equals 90°; the angle of deviation, the difference between the two, then equals 49° 11′. When the refraction which takes place at each surface of a prism is equal, the minimum amount of deviation is present. When the ray is perpendicular to one surface, the angle of incidence at the second surface equals the angle of the prism; the deviation is greater in this case, as all the refraction takes place at one surface. A table of the minimum deviation of prisms will be found on page 20.

Numbering of Prisms.—The designation of prisms by their angular deviation, instead of by their refracting angles, was urged by Dr. Edward Jackson, of Denver, before the Ninth International Medical Congress. Two methods of accomplishing this have been proposed:

Dennett's Method: The Centrad.—Dr. William S. Dennett's calculation has for its base an arc called the *radian*, whose length equals the radius of its curvature. Such an arc equals 57.295°. A prism which produces an angular deviation of the one-hundredth part of this arc is called *one centrad*, denoted by the sign ▽. The deviation of such a prism would, therefore, be 0.57295°. The merit of this method

consists in the uniformity of the deviation, 10 centrads being exactly ten times the deviation of 1 centrad. The deviations are so many hundredths of the radius measured on the arc.

Prentice's Method: The Prism-diopter.—Mr. Charles F. Prentice proposes, as the standard of deviation, a prism which shall deflect a ray of light 1 cm. at a plane 1 meter distant—that is, the hundredth part of the radius measured on the tangent. This he calls the *prism-diopter*, denoted by the sign △. The relative values of the centrad and prism-diopter are recorded in the accompanying table.

There are two practical advantages connected with the method of Mr. Prentice which also can be applied to the centrad. The prismatic deviation of a decentered lens may be very readily found, as Prentice has shown by the following rule: If a lens be decentered 1 cm., the prismatic deviation of the lens will be equal to as many prism-diopters as the number of diopters in the lens. Thus, if a 4-diopter lens be decentered 1 cm., the prismatic deviation will be 4 prism-diopters, or 4 centrads, since centrad and prism-diopter equal each other. The same lens decentered 0.5 cm. would produce 2 prism-diopters or centrads of deviation.

Table of Relative Values of Centrads and Prism-diopters, Prepared by James Wallace

Centrads.	Prism-diopters.	Refracting angle of prism required.
1	1	1.06°
2	2.0001	2.16°
3	3.0013	3.24°
4	4.0028	4.32°
5	5.0045	5.40°
6	6.0063	6.47°
7	7.0115	7.54°
8	8.0172	8.62°
9	9.0244	9.68°
10	10.0333	10.73°
15	15.114	16.1°
20	20.270	21.13°
40	42.288	39.0073°

The prisms represent the minimum deviation with an index of refraction of 1.53.

The relation to the meter angle (see page 45) is also very simple. One-half the interpupillary distance is the sine of the meter angle. The ratio of this to the point of fixation in hundredths gives nearly the number of prism-diopters, or centrads of deviation, embraced in any number of meter angles. For example, if the interpupillary distance is 60 mm., one-half of this is 30 mm., assuming the amount of convergence to be 4 meter angles, 25 cm., or 250 mm., is the distance of the point of fixation. The deviation of the visual line then is 30 in 250, or 12 in 100 = 12 centrads, or 12 P. D. For small arcs the tangent and the sine agree very closely with the arc. Four meter angles of convergence then represent 12 centrads of deviation or 12 prism-diopters.

Rays of Light.—Any luminous point diffuses light in all directions in straight lines called *rays*. As the rays proceed from the luminous source, those which diverge from one another become more widely separated (Fig. 4).

If a circular aperture 1 cm. in diameter be made in a metal plate and a luminous point be placed at different distances from it—for example, at 1 meter and at 10 meters—the rays coming from 10 meters, which pass through the aperture, will be less diverging than those which come from 1 meter. A cone of light will pass through the aperture in each case, but the shape of it will be different according to the distance of the light from the aperture in the screen. When the round hole, 1 cm. in diameter, is 1 meter distant from the point of light, the cone has a base 1 cm. in diameter, and the apex is situated in the luminous point 100 cm. distant. The rays have diverged 1 cm. in traveling 100; the metal plate has cut off all other rays having a greater divergence. If the cone of light passes through the aperture and falls upon a distant wall, the cone will preserve the same proportions—viz., the base will be $\frac{1}{100}$ of the altitude. If the wall be 5 times the distance of the screen from the light, a luminous circle 5 cm. in diameter will be formed upon the wall. If, now, the light is removed to a point 10 meters from the screen (1000 cm.), a cone of light is formed whose base is 1 cm. and whose altitude is 1000. The rays which pass through the aperture have now only $\frac{1}{10}$ of



FIG. 4.—Divergence of rays from a luminous source (Loring).

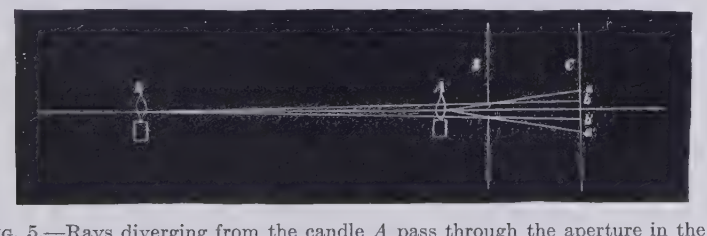


FIG. 5.—Rays diverging from the candle *A* pass through the aperture in the screen *S*, and form the cone of light whose base is the distance *a*–*a'*. Rays from a more distant candle, *B*, having a greater divergence than *b*–*b'*, are intercepted by the screen *S* (Wallace).

the divergence of the rays in the former case; the base of the cone is $\frac{1}{1000}$ of the altitude. The cone of light will now form a circle on the wall 5 meters beyond the aperture, only 1.5 cm. in diameter. If the point of light be at a very great distance, there will be no difference in the size of the luminous circle and the aperture in the screen; the size of the circle remains about 1 cm. on the wall at 5 meters from the screen. The rays, therefore, have a nearly parallel direction. This is shown in Fig. 5.

Rays which enter the pupil of the eye from a point 6 meters distant have so little divergence that they may be considered parallel. The average size of the pupil being 4 mm., the divergence is only $\frac{4}{6000}$. All rays diverging more widely than this are excluded by this width of the pupil.

The relation to the eye of rays diverging from 6 meters or coming from an infinite distance is practically identical, but for lenses of long focal distance and large aperture an infinite distance is required in order to obtain parallel rays. Thus the sun and stars are so remote that the rays coming from them have no appreciable divergence, and they are considered parallel.

Parallel rays must emanate from a distant object. They are brought together by a lens at its principal focus. Conversely, rays which diverge from the principal focus of a lens are parallel to one another after being refracted by the lens.

Divergent rays emanate from an object nearer than infinity. A greater refractive power must be exercised to bring them together at the same distance behind a lens than is required for rays which are parallel; consequently, divergent rays are united at a point farther than the principal focus. The nearer the point of divergence lies to the lens, the farther away from the lens is the point where the rays converge to a focus.

Convergent rays do not exist in nature. Only such rays are convergent which have passed through a convex lens or have been reflected from a concave mirror.

Significance of the Different Rays.—The refraction of the eye is determined by the character which the rays must have in order to be brought to a focus on the retina.

An *emmetropic eye*, with relaxed accommodation, requires rays to be parallel in order that they shall meet on the retina.

A *myopic eye* requires rays to diverge from some near point in order to meet on its retina.

A *hyperopic eye* requires rays which already have convergence to some point in order to unite them on its retina.

An emmetropic eye emits parallel rays.

A myopic eye emits convergent rays.

A hyperopic eye emits divergent rays.

Lenses.—A lens is a portion of glass or other transparent substance bounded by two curved surfaces, or by one curved surface and one plane surface. The curved surfaces are convex, elevated in the center, and thin at the edge; or they are concave, hollowed out in the center and thick at the edge.

A lens may be regarded as a series of prisms with the refracting angles increasing in value from the center toward the periphery (Fig. 6).

In a *convex lens* the bases of the prisms are directed toward the center of the lens, and rays, therefore, are refracted toward the axis which passes through the center. In a *concave lens* the bases of the

prisms are directed away from the center, and rays, therefore, are refracted away from the axis. As the angles increase from the center outward, the peripheral rays will be refracted more than the central rays. The result of this is that in a convex lens the rays after refraction converge to the same point, the increased bending of the more peripheral rays just sufficing to compensate for their greater distance

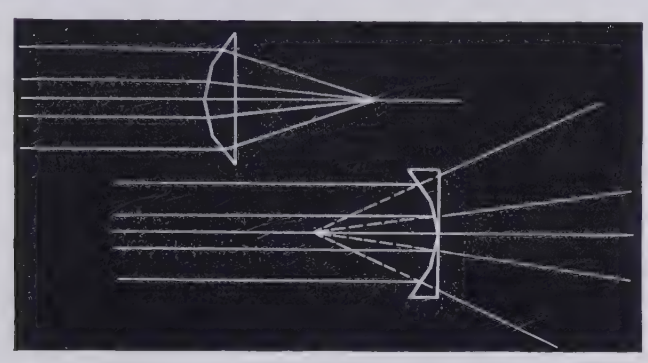


FIG. 6.—Lenses as prisms.

from the axis. In a concave lens the rays diverge more widely as they pass through the peripheral parts of the lens, with the result of making them appear to have diverged from a common point.

Focus of a Convex Lens.—The point to which rays converge after refraction by a convex lens is called its *focus*.

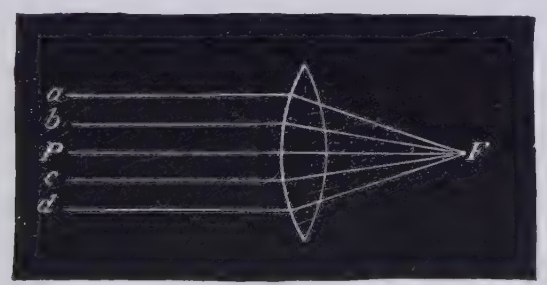


FIG. 7.—Principal focus of a convex lens. The parallel rays *a*, *b*, *c*, *d* are refracted by the lens so as to unite at the point *F* on the axis *P*. The ray *P* undergoes no refraction. *F* is the principal focus.

Principal Focus of a Convex Lens.—The principal focus of a lens is the focus for parallel rays. As only the most distant rays are parallel, never convergent, the principal focus is the shortest focus, unless the lens is combined with another convex lens or concave mirror. Rays diverging from the principal focus of a lens are rendered parallel after passing through the lens, and come to a focus at an infinite distance.

Conjugate Focus of a Convex Lens.—When rays diverge from any point nearer than infinity, they are brought together at a point on the other side of the lens farther than the principal focus. The point from which rays diverge and the point to which they converge are called *conjugate foci*. As the point of divergence approaches the lens the point of convergence recedes; when the point of divergence is at twice the focal distance of the lens, the point of convergence is at an equal distance on the other side. The *conjugate foci* are now equal.

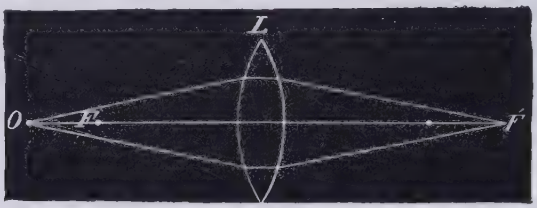


Fig. 8.—Conjugate focus of a convex lens. The two dots in the axis represent the principal foci, one being marked *F*. Rays diverging from *O* converge after refraction to the point *F'*, farther than the principal focus. Rays from *F'* also converge after refraction to *O*. *O* and *F* are conjugate foci.

As the point of divergence approaches still closer the point of convergence is at a greater distance, until, when the point from which the rays diverge is at the principal focus, the rays converge at an infinite distance.

Rays diverging from either of these points converge toward the other. When rays diverge from a point whose distance is equal to or greater than the principal focus, the conjugate focus is *positive*. When the distance is less than the principal focus, the conjugate focus is *negative*.

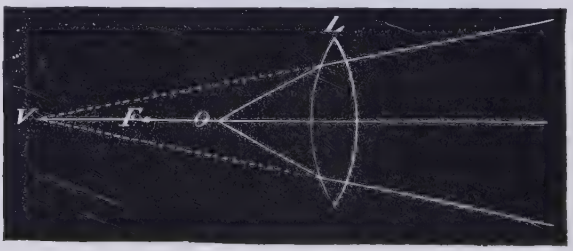


Fig. 9.—Virtual focus of a convex lens. Rays from the point *O*, less than the principal focal distance, diverge after refraction as if they came from the point *V*. *V* is the virtual focus of *O*.

Virtual Focus of a Convex Lens.—When rays diverge from some point nearer to a lens than its principal focus, the rays after refraction still continue divergent. These divergent rays, if traced backward, would meet in a point on the same side of the lens from which they diverged. This point is called a *negative*, or *virtual*, focus, because the rays do not really meet here, but are given a direction by the lens as if they had diverged from this point (Fig. 9). Therefore the point

from which rays diverge and the point to which they converge are *focal points.*

Foci of Concave Lenses.—The foci of concave lenses for parallel or divergent rays are virtual, or negative. They are the points from which the rays seem to diverge after passing through the lens.

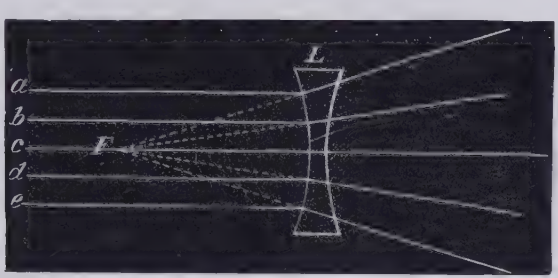


FIG. 10.—Principal focus of a concave lens. Parallel rays *a*, *b*, *d*, *e*, after refraction by the concave lens *L*, are rendered divergent as if they came from the point *F* on the axis *c*. The ray *c* is not refracted. *F*, the principal focus of a concave lens, is virtual.

Principal Focus of a Concave Lens.—When parallel rays fall upon a concave lens they are rendered divergent. If these rays be traced backward, they will seem to have diverged from a point near the lens. This point is the *principal focus* (Fig. 10).

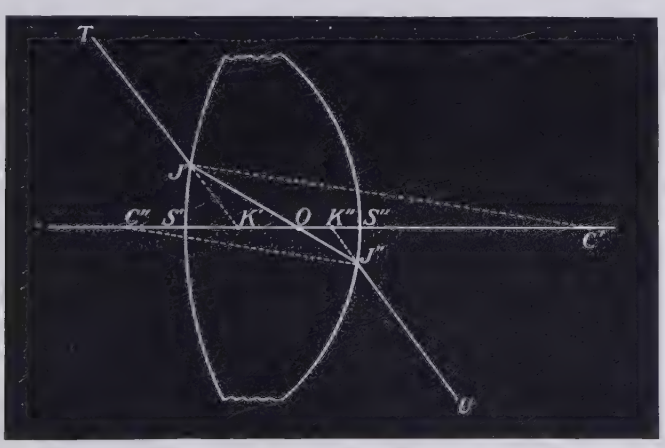


FIG. 11.—*O*, Optical center of lens. The point *C''* is the center of curvature for the surface *S''*. The point *C'* is the center of curvature for the surface *S'*. A ray passing from *C''* to *C'* would be perpendicular to both surfaces. It would pass through without deviation. This ray is called the *axial ray*, or *axis*.

The radii *C''–J''* and *C'–J'*, being parallel, a ray in the lens passing in the direction *J'–J''* must form equal angles at the two surfaces. The point where this ray intersects the axis is the *optical center* (Landolt).

Conjugate foci of concave lenses are also virtual and found in a similar manner.

Formation of Images by a Lens: Optical Center.—In the lens (Fig. 11) the point *O* on the axis is called the *optical center*. Any ray

passing through this point is refracted equally at both surfaces, since it forms equal angles with the radii of the two surfaces. The direction of the ray is, therefore, the same after refraction by the second surface as it was before refraction by the first. For thin lenses it may be said that any ray directed to the optical center passes through without deviation. These rays are called *secondary axes.*

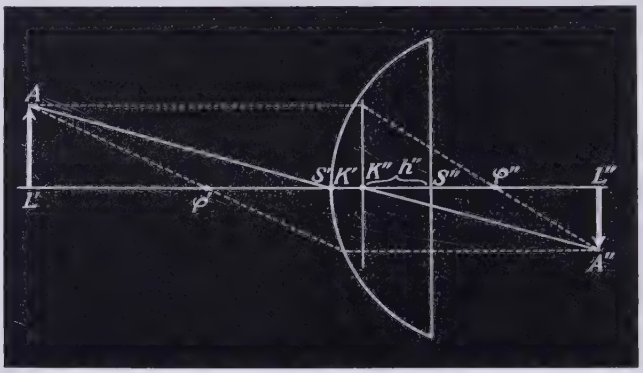


FIG. 12.—Position and size of image formed by convex lens. The ray A, K', from the point A, being directed to the optical center of the lens, continues its course in a parallel direction, K''–A''. Another ray passing from A parallel to the axis L', L'', is refracted through φ'', the principal focus, and, intersecting the ray A–K''–A'', determines the position of the image of the point A. Still another ray passing from A through the anterior principal focus φ', after refraction, is parallel with the axis L'–L'', and meets the other rays in the point A'' (Landolt).

The ray drawn from any point in an object to the optical center of a lens gives the line on which the image of the point is to be found. A ray from the same point in the object, passing parallel to the axis of the lens, would be refracted through the principal focus of the lens, since the principal focus is the focus for parallel rays (Fig. 12).

In order to find the position and size of an image formed by a lens it is only necessary to draw two lines from each extremity of the object:

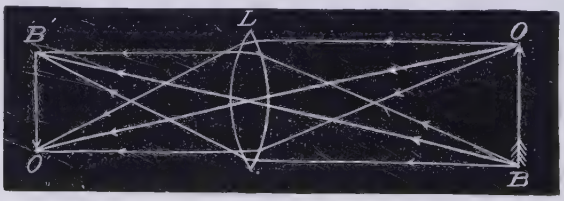


FIG. 13.—Image formed by a convex lens: O–B is the object; O'–B' is the inverted image.

one passes through the optical center of the lens, and the other, parallel with the axis of the lens, would be refracted to the principal focus. The *position of the image* is found at the points where these lines intersect.

The size of the image is proportional to the size of the object as the distance of the image from the optical center is to the distance of the object from the optical center. When the object is situated at a greater

distance from the lens than its principal focus, the image is a real, inverted one.

In the figure (Fig. 13) O–B is the object; the rays diverging from O intersect in O', which is the position of the image of the point O. Similarly the rays from A unite in B', the position of the image of the point B; B'–O' is the image of O–B.

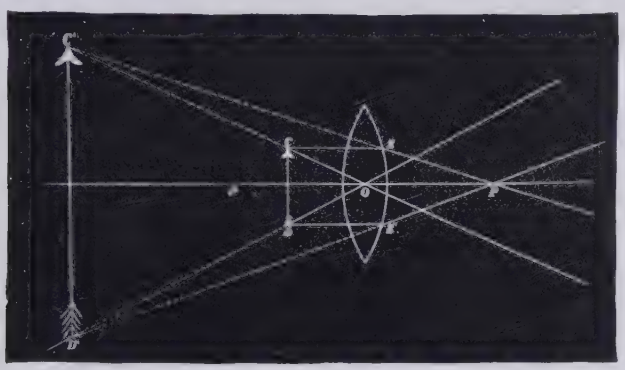


FIG. 14.—Virtual image of a convex lens: C–D is the object; C'–D' is the virtual image, erect and magnified.

When the object is situated nearer to the lens than its principal focus, the image is a virtual, erect one.

The *virtual* image of a *convex* lens appears to be at the point from which the rays refracted by the lens seem to have diverged (Fig. 14). From the point C, of the object C–D, the ray C–S is parallel to the axis. It, therefore, is refracted to the principal focus, P. The ray C–O passes through unchanged. By projecting these rays backward they meet in

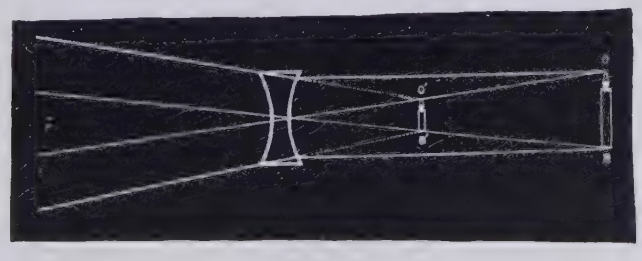


FIG. 15.—Virtual image of a concave lens: O'–B' is the virtual image of the candle, O–B, erect and diminished in size.

C', the image of the point C. The rays from the point D seem to have diverged from D'. An enlarged, *erect* image is thus formed in C'–D'.

The image formed by a *concave* lens is mostly *virtual* and diminished. Two rays, proceeding from a point O, in the object, one parallel to the axis, which seems, after refraction, to have diverged from the principal focus, and is traced backward, and the other, which is directed to the optical center, at their intersection, denote the position of this point in the image (Fig. 15). The enlarged image formed by a convex

lens, and the diminished image formed by a concave lens, as described in the preceding paragraph, are among the most obvious effects of such lenses as they are ordinarily used. It must be remembered, however, that convex lenses are not essentially magnifiers nor concave lenses essentially minifiers, inasmuch as their effect on images depends upon their position with reference to optical systems which they supplement.

Focal Distance of a Lens.—The distance from the optical center of a lens to the focal point is called the *focal distance.*

The length of this depends upon the radii of curvature of the surfaces of the lens and on its index of refraction. Representing the radius by r, the index of refraction of the lens by n, that of air being 1, $F = \frac{r}{2(n-1)}$ is the formula for obtaining the focus of a bispheric convex or concave lens. The formula for a planospheric lens is $F = \frac{r}{n-1}$. The refraction is effected at one surface if the rays are parallel as they enter or pass from the plane surface; otherwise refraction occurs at the plane as well as at the curved surface.

Numeration of Lenses.—The refractive power of a lens is the inverse of its focal distance. If the refractive power of a lens whose focal distance is 1 meter is represented by 1, then a lens whose focal distance is 2 meters has only one-half the refractive power of the first, since the rays are not bent so sharply by the second lens. Again, if a lens bends rays so sharply that they meet the axis at 0.5 meter distance, its refractive power is twice that of a lens of 1 meter focus.

The focus of a biconvex lens (with equal radii), made of glass with an index of 1.50, has the same length as the radius of curvature.

$$F = \frac{r}{2(n-1)} = \frac{r}{2(1.50-1)}$$
$$F = r.$$

Glass used in spectacle lenses has an index of 1.53, consequently—

$$F = \frac{r}{1.06}$$
$$r = 1.06\,F.$$

In the old system the lenses were marked according to their radii of curvature in Paris inches, and the focal distance was somewhat less than the radius of curvature. As all the lenses in use had longer focal distances than 1 inch, they were fractions of the refractive power of a lens of 1 inch focus—viz., $\frac{1}{2}$, $\frac{1}{4}$, $\frac{1}{8}$, $\frac{1}{16}$, etc.

In 1867 Nagel proposed to number lenses by their refractive power. By adopting as a standard a lens of longer focal distance than 1 inch—viz., 1 meter (40 inches)—the greater number of lenses are made multiples of refractive power of the standard, and are based on their focal lengths in meters and fractions of a meter, instead of being based on their radii of curvature.

The term *diopter* was proposed by Monoyer for a lens of 1 meter

focus. A lens of 2 meters focus is only $\frac{1}{2}$ the refractive power, or 0.50 D. The present scale of lenses usually comprises a series from 0.12 to 22 D. Between 0.12 and 1.25 D the lenses have an interval of 0.12 D. From 1.25 to 5 D the interval is 0.25 D; from 5 to 8 D an interval of 0.50 D; from 8 to 18 D an interval of 1 D; and from 18 to 22 D the interval is 2 D. This uniformity in the intervals between the lenses is an important advantage over the old system, in which the lack of uniformity in this respect was a conspicuous feature.

	Number of lens in diopters.	Focal distance in millimeters.	Focal distance in English inches.	Nearest corresponding lens in old system.
Interval of 0.12 D..	0.12	8000	314.96	
	0.25	4000	157.48	144
	0.37	2666	104.99	
	0.50	2000	78.74	72
	0.62	1600	62.99	60
	0.75	1333	52.5	48
	0.87	1143	44.99	42
	1	1000	39.37	36
	1.12	888	34.99	
	1.25	800	31.5	30
	1.5	666	26.22	24
	1.75	571	22.48	
	2	500	19.69	20
	2.25	444	17.48	18
	2.50	400	15.75	16
	2.75	363	14.31	15 or 14
Interval of 0.25 D..	3	333	13.12	13
	3.25	308	12.11	12
	3.50	285	11.25	11
	3.75	267	10.49	10
	4	250	9.84	9
	4.25	235	9.26	8
	4.50	222	8.74	8
	4.75	210	8.29	
	5	200	7.87	
	5.50	182	7.16	7
	6	166	6.54	
Interval of 0.5 D...	6.50	154	6.06	6
	7	143	5.63	5
	7.50	133	5.25	
	8	125	4.92	
	9	111	4.37	4.5
	10	100	3.94	4
	11	91	3.58	3.5
	12	83	3.27	3.25
	13	77	3.03	3
Interval of 1 D....	14	71	2.8	2.75
	15	66	2.64	
	16	62	2.44	2.5
	17	59	2.32	2.25
	18	55	2.17	
	20	50	1.97	2
Interval of 2 D....	22	45	1.79	

To find the focal length of any lens in the dioptric system divide 1 meter, or 100 cm., by the number of diopters: thus the focal length of a lens of 5 D is $\frac{100}{5} = 20$ cm.

In the old system the lenses are ground with a radius of curvature in Paris inches. The focal length is almost exactly the same in English inches as the radius of curvature is in French inches. The English inch = 25.4 mm.; the French inch = 27.07 mm.; $25.4 \times 1.06 = 26.92$.

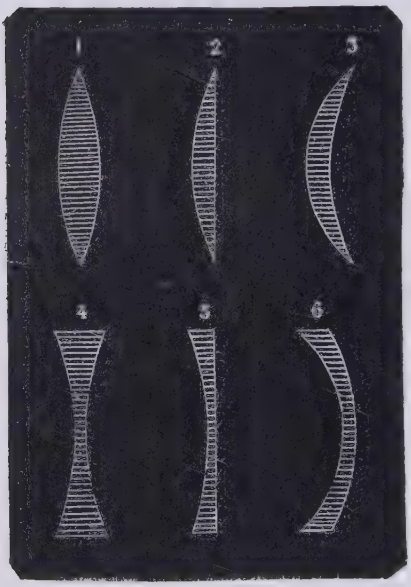


FIG. 16.—1. Biconvex lens. 2. Planoconvex lens. 3. Concavoconvex lens, convergent meniscus. 4. Biconcave lens. 5. Planoconcave lens. 6. Convexoconcave lens, divergent meniscus.

In column three of the table the focus is given in English inches, as it is customary to compare the French lenses with the diopters by their focal length in English inches. A lens of 1 diopter has a focal length of 39.37 English inches. There is no lens in the old system which corresponds to it exactly. The nearest equivalent would be a lens of 40 inches.

The lenses used for spectacles are spheric and cylindric.

Spheric Lenses.—A spheric lens is represented by a section of a sphere, or of two sections of a sphere placed together by their plane surfaces. Light passing through a spheric lens is refracted equally in all planes.

Cylindric Lenses.—A cylindric lens is a section of a cylinder parallel to its axis. Light passing through a cylindric lens is not refracted in a plane parallel to its axis, but in a plane perpendicular to the axis; rays are rendered convergent or divergent according as the cylinder is convex or concave (Figs. 17, 18).

Convex lenses are designated +; concave lenses, —.

Toric Lenses.—A solid developed by the revolution of a circle about any axis other than its diameter is known as a *torus*. A *toric lens* may be described as one which is cut from a toric surface by a plane parallel to its axis of development. The optical centering of such a lens requires that both its centers, the center of its circle and the center about which in its development the circle revolves, shall be on the axis of the system (W. S. Dennett). With the toric lens the angle of distinct view is increased, but a certain amount of astigmatism remains in the greater number of powers. To eliminate this astigmatism of oblique pencils of light M. von Rohr has developed a lens which it is claimed "reproduces any given definite point of an object

as a distinct point in the image, that is, a lens which is corrected for astigmatism over the entire field of vision in all powers." To this lens the name *Punktal* has been given.

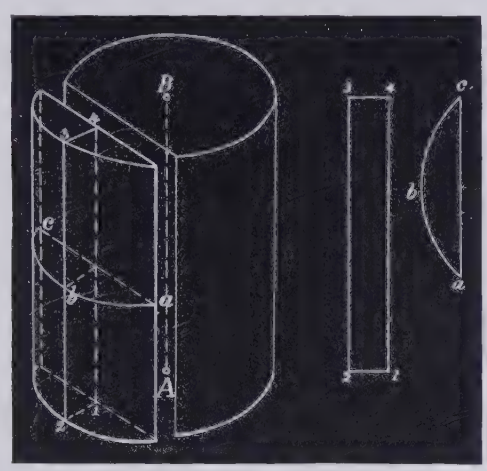


FIG. 17.—Convex cylindric lens, formed by a section of a cylinder parallel to its axis, which acts like a plane lens (*1*, *2*, *3*, *4*), in a direction parallel to the axis of the cylinder (*A*, *B*), and like a convex lens (*a*, *b*, *c*), in a direction perpendicular to the axis.

Combination of Lenses.—If two or more lenses are placed together, for example, + 2 diopters, + 3 diopters, and + 4 diopters, the

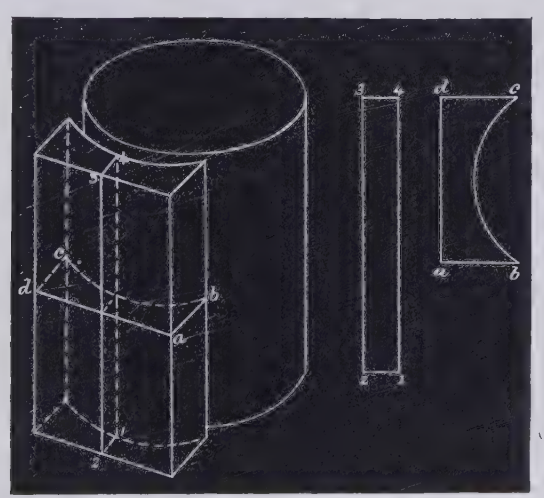


FIG. 18.—Concave cylindric lens, formed from a solid cylinder; in a plane parallel to the axis it acts like a plane lens (*1*, *2*, *3*, *4*), but in a plane perpendicular to the axis like a concave lens (*a*, *b*, *c*, *d*).

combination forms a dioptric power equal to their sum—viz., 9 diopters; such a combination has, if composed of thin lenses, a focal distance of

$\frac{100}{9}$ = 11 cm. If these lenses are placed at their focal distance from an object, the rays coming from the object, after passing through the lenses, are parallel.

Two or more concave lenses placed together likewise produce a dioptric effect equal to their sum.

Combination of Convex and Concave Lenses.—If a concave and a convex lens of equal strength are placed together, they will neutralize each other so exactly that a distant object viewed through them will appear neither enlarged nor diminished, and there will be no prismatic deviation on gently shaking the lenses in a direction parallel to the surface. Should they be unequal in strength, the object (the edge of a window frame is suitable), under similar conditions, will be displaced toward the center of the lens if the concave is stronger, and away from the center if the convex is stronger. The value of the combination will be the difference between the strength of the two. For instance, a + 3 diopter and a — 2 diopter equal + 1 diopter; a + 2 diopter and a — 4 diopter = — 2 diopter.

A — 2-diopter lens gives to parallel rays a direction as if they came from a point 50 cm. away. Conversely, rays diverging from any near point may be represented by a concave lens, the principal focus of which equals that distance. Let rays, for example, diverge from a point 15 cm. away; they evidently are similar to parallel rays which have passed through a concave lens of 15 cm. focal distance, $\frac{100}{15}$ = 6.66 diopters.

If it is desired to find the conjugate focal distance of any lens for rays which diverge from 15 cm., 6.66 should be subtracted from the dioptric power of the lens; the remainder gives a lens the focal distance of which is the conjugate desired. If it is desired to find the conjugate focal distance of a 12-diopter lens for rays which diverge from 15 cm., 6.66 should be subtracted from 12 = 5.33 diopters; 18.8 cm. is the conjugate focal distance.

Combination of Cylindric Lenses with Spheric Lenses.—A cylindric lens is curved only in the direction *perpendicular to its axis;* rays which enter the lens are refracted in this plane to the focus of the lens exactly as in the case of a spheric lens.

In the opposite direction, that is, *parallel to its axis*, the surface of a cylindric lens is flat; rays entering are not refracted in this plane, but pass through unchanged. The effect of a cylindric lens placed in front of the eye is to increase or diminish its refraction in the direction at right angles to its axis, but in the opposite direction the refractive power is unchanged (see Figs. 17, 18).

A convex 4-diopter cylindric lens, with its axis in a vertical direction (written + 4 D cyl., axis 90°), increases the refraction in the horizontal direction 4 diopters, but does not alter the refraction in the vertical direction. The horizontal plane is expressed by the term *horizontal meridian;* the vertical plane by the term *vertical meridian.*

A concave cylindric lens of 4 diopters, with its axis horizontal (written — 4 D cyl., axis 180°), diminishes the refraction of the vertical me-

ridian 4 diopters, but does not affect the refraction of the horizontal meridian.

A convex lens of 3 diopters, combined with a convex cylindric lens of 2 diopters, with its axis vertical (written + 3 D ⌒ + 2 D cyl., axis 90°), adds to the horizontal meridian + 5 diopters, but to the vertical meridian only 3 diopters.

The combination of a convex spheric lens with a concave cylindric lens has the following effect: In the direction parallel to the axis of the cylinder the combination equals the full refraction of the spheric; in the direction at right angles to the axis of the cylinder the refraction is equal to the difference between the two lenses. If the convex spheric is stronger than the concave cylinder, the difference is still represented by a convex glass. For example, + 2 D sph., ⌒ — 1.50 D cyl., axis 180° = + 0.50 D sph., ⌒ + 1.50 D cyl., axis 90°, because + 2 D in the meridian of 180° is not diminished, but in the meridian of 90° it is reduced to + 0.50 D. Now, + 0.50 D sph. produces this amount of refraction at 90°, and supplies + 0.50 D of the requisite + 2 D at 180°, leaving + 1.50 D to be supplemented by a cylindric lens with its axis at 90°.

In place of writing + 2 D sph., ⌒ — 1.50 D cyl., axis 180°, a more simple expression would be + 0.50 D sph., ⌒ + 1.50 D cyl., axis 90°.

Where, however, the concave cylindric lens is stronger than the convex spheric, the difference is represented by a concave lens, thus + 3 D sph., ⌒ — 6.50 D cyl., axis 180°, signifies in the horizontal meridian convex 3 D, and in the vertical meridian concave 3.50 D. It is necessary to combine a convex with a concave lens in order to obtain this effect. The refractive power of this combination can be expressed in three different ways:

+ 3 D sph., ⌒ — 6.50 D cyl., axis 180°.
— 3.50 D sph., ⌒ + 6.50 D cyl., axis 90°.
+ 3 D cyl., axis 90°, ⌒ — 3.50 D cyl., axis 180°.

In the first combination + 3 D sph. gives the + 3 D necessary for the horizontal meridian, but increases the refraction of the vertical meridian 3 D instead of diminishing it; therefore the — 6.50 D cyl., axis 180°, expends 3 D of its refractive power in neutralizing the effect of the + 3 D sph., and with the remainder diminishes the refraction of the vertical meridian 3.50 D.

In the second combination, — 3.50 D sph., ⌒ + 6.50 D cyl., axis 90°, the concave spheric lens diminishes the refraction of the vertical meridian 3.50 D, but also diminishes the refraction of the horizontal meridian 3.50 D; as this already requires + 3 D, we must add + 3.50 D more to compensate for the concave spheric, making + 6.50 D cyl., axis 90°.

In the third combination, + 3 D cyl., axis 90°, ⌒ — 3.50 D cyl., axis 180°, + 3 D cyl., axis 90° increases the refraction of the horizontal meridian without altering the refraction of the vertical meridian, and the — 3.50 D cyl., axis 180° diminishes the refraction of the vertical meridian without affecting the refraction of the horizontal meridian.

With the combination of a convex spheric and cylindric lens, *e. g.*, + 3 D sph., ⌒ + 2 D cyl., axis 90°, a concave 0.50 D cylinder with its axis at right angles to the axis of the convex cylinder, in this case at 180°, diminishes the refraction of the vertical meridian 0.50 D, the combination then equals + 2.50 D in the vertical meridian and + 5 D in the horizontal = + 2.50 D sph., ⌒ + 2.50 D cyl., axis 90°.

A convex cylinder + 0.50 D added to the same combination, with its axis at right angles to the axis of the first cylinder, that is, + 0.50 D cyl., axis 180° with + 3 D sph., ⌒ + 2 D cyl., axis 90°, increases the refraction in the vertical meridian + 0.50 D. The combination then equals + 3.50 D in the vertical meridian, + 5 D in the horizontal. This is obtained by + 3.50 D sph., ⌒ + 1.50 D cyl., axis 90°.

Visual Angle.—The apparent size of an object depends upon the size of the *visual angle.*

The visual angle is the angle formed by the lines drawn from the two extremities of an object to the nodal point of the eye. The *nodal point* of the eye is analogous to the optical center of a lens. It is situated 15 mm. in front of the retina and 7 mm. behind the cornea. Rays directed to this point pass through without deviation.

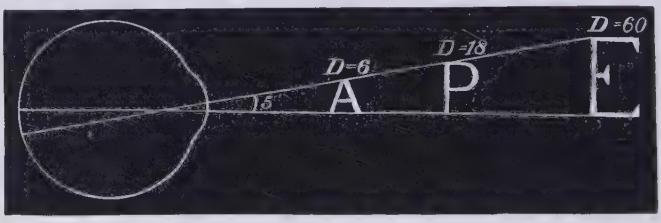


Fig. 19.—The visual angle.

As the rays directed to the nodal point of the eye are not refracted, but continue the same course until they strike the retina, if lines are drawn from the extremities of an object through the nodal point of the eye, and continued until they fall upon the retina, the size of the retinal image of the object is obtained.

The figure shows that the object, in order to subtend the same angle, must be larger the farther it is removed from the eye. The letter *A*, seen clearly at 6 meters, would have to be three times as large in order to be seen distinctly at 18 meters, and ten times as large in order to be seen clearly at 60 meters. The visual angle in the three instances remains the same.

Retinal Image in Emmetropia.—In the emmetropic eye the *nodal point* is situated 7 mm. behind the cornea and 15 mm. in front of the retina. The size of the retinal image is to the size of the object as the distance from the retina to the nodal point (15 mm.) is to the distance from the nodal point to the object. Therefore, if an object is situated at 1 meter distance (1000 mm.), its image will be $\frac{15}{1000}$ of the size of the object.

Retinal Image in Ametropia.—In the hyperopic eye, the axis of which is shorter than that of the emmetropic eye, the retina is situated nearer the nodal point; the image is, therefore, smaller. In myopia the axis of the eye is longer; the retinal image is, therefore, larger.

Visual Acuteness; Limit of Perception.—An object 1 cm. in size, placed 1 meter distant from a normal emmetropic eye (that is, an eye without any error of refraction), is plainly visible. If this object is moved farther and farther away, it forms a progressively smaller visual angle, until a point is reached beyond which it cannot be perceived, owing to the diminutive size of the visual angle. The *limit of perception* has now been reached.

The angle which the object subtends at this distance from the eye represents the maximum *acuteness of vision*. An object twice the size would be seen distinctly at twice this distance. An object one-half the size could not be distinctly seen at more than half this distance. In general terms the size of the object denoting the acuteness of vision is always proportional to the distance.

Normal Acuteness of Vision.—Snellen determined the normal acuteness of vision to be the power of distinguishing letters subtending an angle of 5′. These letters are formed of strokes whose width is $\frac{1}{5}$ the size of each letter; consequently they are seen under an angle of only 1′. The openings in the letters and the spaces between contiguous strokes, as nearly as possible, are made to conform to the same angle.



FIG. 20.—Two of Snellen's test-types.

The relation of the size of the letter to the distance at which it should be discerned by a normal eye is expressed by twice the tangent of half the angle of 5′ = 0.001454. The size of a letter the perception of which constitutes normal vision at a given distance may be obtained by multiplying the distance by 0.001454. At the distance of 1 meter the size of this standard letter is 1.45 mm. (0.001454 × 1000 mm.). At a distance of 6 meters the size of the letter required is 8.7 mm. (1.454 × 6). The size of the retinal image of a standard letter of 6 meters = $\frac{15}{6000}$ of 8.7 = 0.02175 mm., and the strokes, or openings, being $\frac{1}{5}$ the size, have an image of 0.00435 mm. A large number of people, after correction of their ametropia, have a visual acuteness of 1.25 of normal, and, therefore, letters constructed on an angle of 4′ have been used for testing visual acuteness. The retinal images of the strokes of such letters are $\frac{4}{5}$ of 0.00435 = 0.00348 mm. The size of the cones of the macular region varies from 0.0033 to 0.0036 mm., showing a most interesting relation between the limit of perception and the anatomic structure of the retina.

ACCOMMODATION

Mechanism of Accommodation.—The eye cannot adapt itself for the perception of objects situated at different distances by increasing

the length of its axis, but only by increasing the refractive power of its lens. Rays diverging from near objects are thus brought to a focus at the same distance as the rays diverging from remote objects. This power the eye possesses of adapting its refraction for different distances is called *accommodation*, and the change required in its optical adjustment is effected by the ciliary muscle in the following manner: The ciliary muscle, which lies between the sclera and the ciliary processes, and which is attached posteriorly to the choroid tract by fibers known as the *tensor choroideæ*, contracts. This contraction draws forward the anterior part of the choroid and the posterior part of the ciliary body, to the ciliary processes of which is attached the suspensory ligament of the lens or zonula of Zinn. Hence the zonula is relaxed, and the tension which it has exerted on the lens capsule is removed. The crystalline

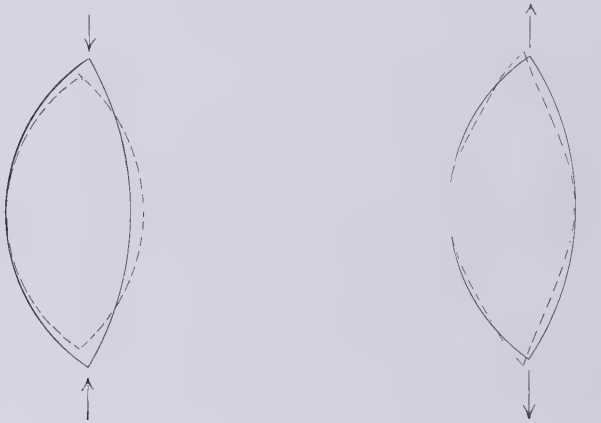


FIG. 21.—*A*, Accommodation according to Helmholtz. The dotted line represents the thicker form assumed by the lens when the traction of the zonula is diminished by the contraction of the ciliary muscle. *B*, Accommodation according to Tscherning. The unbroken lines show the lens at rest. The dotted lines show the change occurring during accommodation, supposed to be due to the traction of the zonula being increased by the contraction of the ciliary muscle (Cutler).

lens, a soft and elastic body, thus freed from compression, tends to assume a spheric shape, bulges forward, and becomes more convex. It has, in effect, added to its anterior surface another convex lens. As the ciliary muscle contracts more vigorously, this added convex lens becomes stronger. This is the Helmholtz theory, and attempts to disprove it have not been successful, as has been shown by Gullstrand and by C. von Hess.

The passive expansion of the elastic crystalline lens when the pressure of the suspensory ligament is relaxed, is denominated by Fuchs *physical accommodation;* the active contraction of the ciliary muscle whereby the relaxation of the zonula is effected, followed by this passive expansion of the lens he names the *physiologic accommodation.*

Tscherning holds a different view of the mechanism of accommodation, thus expressed by Colman Ward Cutler: Accommodation does

not depend on a relaxation of the zonula of Zinn, but on its tension through the agency of the ciliary muscle, whereby the peripheral portion of the lens is flattened and the curve of the anterior surface, from an approximately spheric, approaches a hyperboloid form. Investigations indicate that this theory, thus briefly summarized, is not correct and that the explanation of the mechanism of accommodation given by Helmholtz should be retained, even though, as Duane points out, the Helmholtz theory does not elucidate all the phenomena of presbyopia.

Exercise of the Power of Accommodation.—If an emmetropic individual wishes to see an object situated, for example, 25 cm. distant, he must increase his accommodative power to such a degree that in effect he adds to his crystalline lens another lens of 4 diopters—*i. e.*, one having a focal length of 25 cm. Rays diverging from 25 cm. are thus given a parallel direction and are brought to a focus on the retina by the original refractive power of the eye.

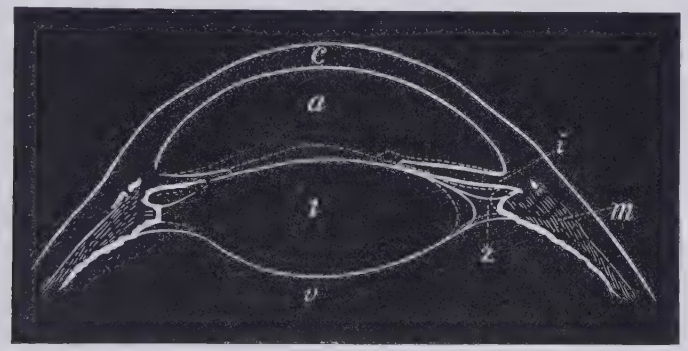


FIG. 22.—Increased convexity of the lens during accommodation. The solid white outline of the lens, *l*, shows its form when relaxed. The dotted line shows the increased curvature of the anterior surface during accommodation, and its advancement forward into the anterior chamber, *a*. *Z* is the suspensory ligament; *m*, the ciliary muscle; and *i*, the iris. (Landolt.)

The degree of accommodation varies according to the distance of the object; it is not possible for an eye to be adapted for two different distances at one time. By means of the accommodation the eye is adjusted for all distances between its farthest and nearest point of distinct vision.

The **far point** of an eye, *punctum remotum*, is the point from which come rays having the least divergence, or toward which go rays having the greatest convergence that allows their focusing on the retina. From this point rays are focused on the retina with the ciliary muscle entirely relaxed, the refraction of the eye being at its minimum, *R*. This point, or its distance from the eye, is designated *r*.

The **near point** of an eye, *punctum proximum*, or *p*, is the point from which come the most divergent rays that can be focused on the retina. These are focused with the ciliary muscle contracted to its fullest extent, and the eye in its condition of the maximum refraction,

expressed by P. The space lying between r and p is called the *region of accommodation*. The extent of this space, however, does not supply a measure of the amount of work of which accommodation is capable which is determined by the *amplitude of accommodation*.

This **amplitude of accommodation,** likewise denominated the *power* or *range of accommodation*, is the difference between the refractive power of the eye accommodated for its far point and accommodated for its near point. This is expressed by A. $A = P - R$.

As the refractive power is the inverse of the focal distance, the refractive power of the eye, when accommodated for its far point r, is $R = \frac{1}{r}$. If we express the value of r in meters, we shall then have the refractive power of the eye expressed in diopters, a diopter being a lens of 1 meter focus. If $r = 1$ meter, $R = \frac{1}{1} = 1$ diopter $= 1$ D. If r is infinitely distant, $R \frac{1}{\infty} = 0$.

In the same manner $\frac{1}{p} = P$, the refractive power of the eye when accommodated for its nearest point. If we obtain the value of p in centimeters and wish to know how many diopters it equals, we must divide 100 by the number of centimeters equal to p. Let $P = 10$ cm., then $P = \frac{100}{10} = 10$ D. If p is expressed in fractions of a meter, we obtain the same result: by dividing 1 by the value of p, in meters, 10 cm. $= \frac{1}{10}$ of a meter. $P = \frac{1}{\frac{1}{10}} = 10$ D, or, in decimals, $1 \div 0.1$ meter $= 10$ D—that is, in order to focus rays from 10 cm., we require 10 times as much accommodation as is necessary to focus rays from 1 meter, and since an eye adapted to a distance of 1 meter exerts 1 diopter of accommodation at a distance of $\frac{1}{10}$ meter, or 10 cm., it must exert 10 diopters of accommodation.

To find the amplitude of accommodation we must first determine the *far point*. This is accomplished by means of test-letters held in front of the patient. If the patient has maximum acuteness of distant vision, r is infinite [when $R = \frac{1}{\infty} = 0$] or negative. If vision is less than normal at 6 meters, but is normal at 1.5 meters, $r = 1.5$ meters; R then $= \frac{1}{1.5} = 0.66$ D. If distant vision becomes or remains distinct when a convex glass of 2 D is placed before the eye, then $R = -2$ D; that is, the far point of such an eye is negative, a point behind the retina toward which rays converge. This condition is further discussed on page 133.

The *near point* is usually found by gradually approaching a card containing fine print until the nearest point from the eye at which it still remains distinct is reached. The distance of this point from the

anterior surface of the cornea is measured. For this purpose large print may be reduced by photolithographing, so as to subtend the standard angle of 5′ at a distance of 25 cm. or less, and is usually arranged on suitably shaped cards. According to Duane, the best test object for practical purposes is a simple engraved line 0.2 mm. thick and 3 mm. long, which, when brought within the near point, blurs slightly and then doubles. In making his estimates he prefers to reckon from the anterior focus of the eye—*i. e.*, from a point 13 mm. in front of the cornea.

The *formula for obtaining the amplitude of accommodation* is $A = P - R$. If p is at 20 cm., $P = \frac{100}{20} = 5$ D, and r is at infinity, $R = 0$, then $A = P = 5$ D. This is the case in emmetropia.

If p is at 10 cm., $P = \frac{100}{10} = 10$ D, and r is at 25 cm., $R = \frac{100}{25} = 4$ D, then $A = 10\text{ D} - 4\text{ D} = 6$ D. This is the case in myopia of 4 D P is greater than A.

If p is at 50 cm., $P = \frac{100}{50} = 2$ D, and r is negative, — 25 cm. $R = \frac{100}{-25} = -4$ D. $A = 2 - (-4) = 2 + 4 = 6$ D. This is the range of accommodation in a hyperope of 4 D, and equals the sum of P and R.[1]

The near point is closer to the eye in young life, while the lens is soft; as age advances the lens becomes harder and the near point gradually recedes until, at about the age of seventy, the near point has reached infinity, and p and r then coincide, and there is no range of accommodation.

TABLE OF THE RANGE OF ACCOMMODATION

10 years	14	diopters	p =	cm.
15 years	12	"	p = 8.3	"
20 years	10	"	p = 10	"
25 years	8.5	"	p = 12	"
30 years	7	"	p = 14	"
35 years	5.5	"	p = 18	"
40 years	4.5	"	p = 22	"
45 years	3.5	"	p = 28	"
50 years	2.5	"	p = 40	"
55 years	1.75	"	p = 55	"
60 years	1	"	p = 100	"
65 years	0.75	"	p = 133	"
70 years	0.25	"	p = 400	"
75 years	0	"	p = ∞	"

[1] p refers to the distance of the near point in centimeters. P refers to the refractive power of the eye in accommodation for p. r refers to the distance of the far point. R refers to the refraction of the eye when accommodated for r.

Although *range of accommodation* and *amplitude of accommodation* are often employed as interchangeable terms, the former, that is, the distance between the near point and the far point, is not always the same for a given amplitude.

The *power of accommodation* is nearly constant for the same age, so that if p is nearer than it should be, myopia may be suspected, or if it is farther away than the average, hyperopia (Fig. 23). For this purpose the table on page 39 is often used, which records the average of P in diopters and p in centimeters for the different ages. (Compare with Fig. 23.)

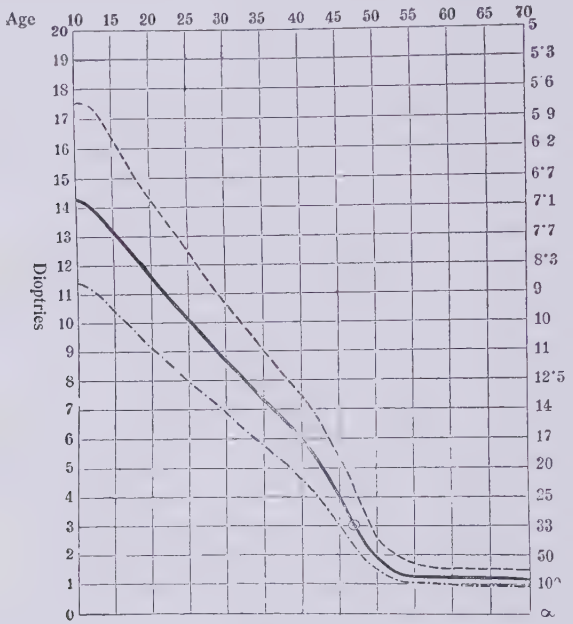


FIG. 23.—Range of monocular accommodation at different ages. The numbers on the left hand give the strength of the lens, which placed before the emmetropic eye at a distance of 13 mm. from the apex of the cornea (*i. e.*, placed at the anterior focus of the eye), can replace the accommodation of the eye at the given age, and hence is equivalent to the accommodation, so far as regards the increase of refractivity in the eye which the latter produces. These numbers, therefore, give the range of accommodation of the eye. The right-hand numbers give the focal distances of these lenses (in centimeters). The distance of the near point from the apex of the cornea is found by adding to the focal strength of the lens its distance from the cornea (*i. e.*, 13 mm.). Obviously, no single observer can follow the progress of the range of accommodation from youth to age in one and the same individual. The progress of accommodation can, therefore, be found only by determining it in a large number of persons with normal eyes at different ages, and taking the mean of the observations. This is shown by the continuous line, which indicates the mean position of the near point (without reckoning in the 13 mm.) of an emmetropic eye at different ages. The upper and lower dotted lines give the least and the greatest distance of the near point that has been found in the individual cases. They accordingly show the limits within which the position of the near point can still be regarded as normal. The ring denotes the limit of presbyopia as conventionally set. (After Donders, amended and described by Duane.)

Duane and J. B. Thomas, in an attempt to determine the normal range of accommodation, have reached conclusions somewhat at variance with those of Donders, which are usually recorded. The accommodation in childhood and youth they found to be not so high as he states. The accommodation does not decrease year after year by

any steady sweep, inasmuch as it may remain unchanged for years at some periods of life. After fifty-one the accommodation remains nearly constant, diminishing only 0.50 D in ten years. Duane's researches are calculated to fix not only the mean range of accommodation, but also the upper and lower limits at each age.

Binocular Accommodation.—According to Duane's researches binocular accommodation is higher than the monocular, the excess being regularly equal to 0.6 D or more below the age of seventeen; 0.5 D from eighteen to thirty-one; 0.4 D from thirty-two to fifty-three, and 0.3 D for ages over fifty-three. For individual cases the difference may be greater.[1]

Abnormal Accommodation.—Failure of accommodation due to age is termed *presbyopia* (see page 161). The chief anomalies of accommodation, adopting and quoting Duane's classification, are *insufficiency of accommodation* (see also page 132), in which the accommodation is constantly below the lower limit; *ill-sustained accommodation*, in which accommodation is normal in amount, but soon gives out; *inertia of accommodation*, in which difficulty in changing from one accommodative state to the other is experienced; *inequality of accommodation* (see also page 163), in which the accommodation in the two eyes is not the same; and *excessive accommodation* in which the accommodation is persistently above the normal limit.

Insufficiency of accommodation, according to Duane, usually occurs, and it is not infrequent between the ages of eighteen and forty-five. Theoretically, it is due to undue rigidity of the lens (*lenticular hypocyclosis* according to Duane) or weakness of the ciliary muscle (*ciliary hypocyclosis*, according to Duane), and may be associated with an undue effort of convergence, that is, convergence excess (see page 639), but also and more frequently with convergence insufficiency (see page 640). The main symptom is *asthenopia*, and prominent causes are various toxemias due to tuberculosis, intestinal disorders, nasopharyngeal, tonsillar and dental disease, hypopituitarism, neurasthenia, overwork, eye strain, and vascular hypertension. In the author's experience early arteriosclerosis is a common cause of this condition. Evidently the remedies are removal of the cause, suitable correcting glasses, with an addition for reading to correct the unnatural presbyopia, and, in the author's experience, the instillation of a weak eserin or pilocarpin solution (of eserin salicylate gr. $\frac{1}{4\,0}$–f ʒj (0.0015 gm.–30 c.c.); of pilocarpin hydrochlorid, gr. $\frac{1}{2\,0}$–f ʒj (0.003 gm.–30 c.c.). Duane recommends in the treatment of *ciliary hypocyclosis* training convergence and direct training of the accommodation accomplished by focusing on the accommodation line (page 39) with one eye and then with the other, and finally with both eyes several times a day. Unequal accommodation, non-pathologic in origin, may give rise to much discomfort, especially when the presbyopic age is approached or reached, and must be recognized in the correcting glasses (see also page 163).

[1] For an important investigation by Duane of monocular and binocular accommodation see Amer. Jour. Ophth., 1922, vol. 5, 865.

Angle Gamma; Angle Alpha.—The eye, in looking at any object, is directed forward in such a manner that the image is formed on the *macula lutea.* The eye is now said to "fix" or fixate the object. A line drawn from the object thus fixated to the macula lutea is called the *visual line,* or *visual axis.*

The point about which the eye revolves, in order to be brought into this position, is called the *center of rotation,* and has its position 14 mm. back of cornea. The line which connects the object with the center of rotation is designated the *line of fixation.*

The *optic axis* is an imaginary line passing through the center of the cornea and lens and the point of rotation to the posterior pole of the eye—*i. e.,* a point usually between the macula and optic papilla.

If the macula lutea coincided with the posterior extremity of the optic axis, the visual line, line of fixation, and optic axis would also coincide. Generally, this coincidence does not exist. In emmetropia and hyperopia the optic axis passes to the inner side of the macula lutea, and the visual line and line of fixation then form angles with the optic axis. In Fig. 24 A–A' is the optic axis passing through the center of the cornea, C, the nodal points of the eye, K'–K'', and the center of rotation, M. O–F is the visual line connecting the object, O, with the *fovea,* F. O–M is the line of fixation, drawn from O to the center of rotation, M. The eye, in order to fix O, has its optic axis, A–A', deviated outward. The angle formed by the line of fixation, O–M, with the optic axis A–A', is called the *angle gamma,* γ, or the angle formed by the visual line with the optic axis may be considered as the angle gamma.

The significance of this angle is that a person, while really fixating an object, seems to have a divergence of the visual lines—divergent squint. In estimating the degree of a divergent strabismus it is necessary to consider the value of this angle. The amount of the angle gamma is usually 5°, but it may reach as much as 10°. When the anterior extremity of the visual line passes to the inner side of the optic axis, the angle gamma is positive, or +; this is the usual condition in emmetropia and hyperopia. The convergence of the visual line exceeds the convergence of the optic axis by the amount of this angle. When the visual line coincides with the optic axis, there is no angle gamma. The visual line in high myopia sometimes passes to the outer side of the optic axis. The eyeball must then be deviated inward in order to fix on the object. This produces the effect of a convergent squint. It must be distinguished from squint, and if convergent strabismus also exists, the value of this angle must be deducted from the apparent squint. In this latter form of the angle gamma, where the anterior extremity of the visual line passes to the outside of the optic axis, the angle is negative, or —. The convergence of the visual line is less than the convergence of the optic axis by the amount of this angle.

The amount of this angle may be measured by placing the patient before the perimeter as if his field were to be taken (see page 631). The eye is fixed on the central point, and a lighted candle is moved

along the arc in a horizontal direction until its reflection is obtained from the portion of the cornea corresponding to the center of the pupil. The position of the candle flame may now be read from the arc in degrees, and represents the size of the angle gamma.

The apex of the cornea does not generally coincide with the center of the cornea, but is displaced laterally. The major axis of the corneal

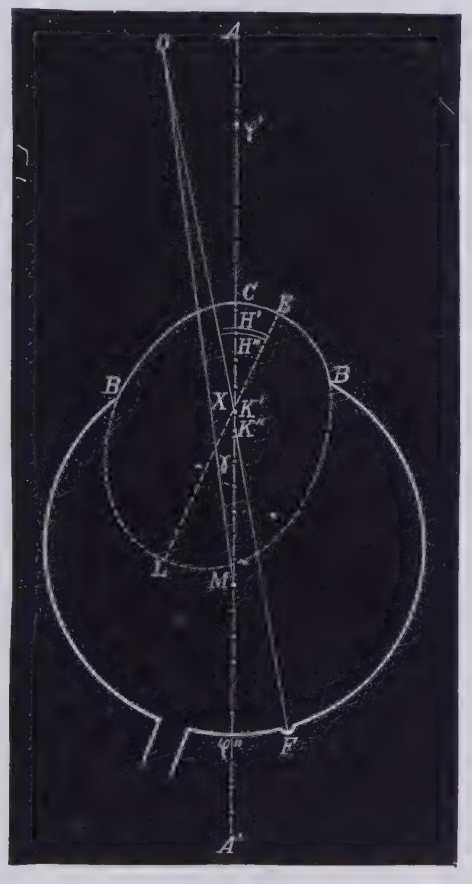


FIG. 24.—Angle alpha and angle gamma: *A–A'*, Optic axis; *O–F*, visual line; *O–M*, line of fixation; *E–L*, major axis of corneal ellipse. The line of fixation does not correspond with the optic axis, but forms the angle *O–M–A*, angle gamma nearly equal to the angle *O–X–A*, formed by the visual line with the optic axis. *O–X–A* may be considered as the angle gamma. The visual line does not pass through the summit of the corneal curve, *E*, but forms with the axis of the cornea, *E–L*, the angle *O–X–E*, the angle alpha (Landolt).

ellipse, represented in the figure by *E–L*, therefore forms an angle with the visual line. The *angle alpha* is the angle formed by the visual line with the major axis of the corneal ellipse. It is *positive* when the major axis of the cornea passes to the outer side of the visual line; if the corneal axis passes to the inner side of the visual line, the angle alpha is *negative*.

In the figure the angle *O–X–A* is the *angle gamma;* the angle *O–X–E* is the *angle alpha.*

From what has been said it will be seen that the visual line is a secondary axis to the optical system of the eye. The oblique position of the refracting surfaces to the visual line may be the cause of an increased refraction in the horizontal meridian constituting astigmatism.

CONVERGENCE

In the visual act of one eye the sensation conveyed to the brain is projected outward over the same course by which it arrived—that is, the object is referred to a position in the field of vision which it actually occupies. If the projection outward of the images of the two eyes is such that they overlie each other, the person will have single vision; if, however, they are projected in different positions, double vision is the result.

The images are projected in different positions when they are not formed on *identical points* of the two retinas. The *fovea centralis* being the most sensitive portion of the retina, the eye is naturally so directed toward an object that the image is formed upon it. The eye is then said *to fix,* or *fixate,* the object. The foveas of the two eyes are identical points, and images formed on them are projected outward so as to overlie or fuse into each other; points at a corresponding distance to the right of each fovea, or to the left, or upward or downward, are also identical, and images formed on them produce but a single impression. Objects in the field of vision to the right of the point of fixation form a retinal image to the left of the fovea. Objects to the left of the point of fixation form an image to the right of the fovea (see Figs. 263, 264). All images formed on the retina to the right of the fovea are projected outward to the left. Those formed on the left of the fovea are projected to the right; in the same way those formed on the upper part of the retina are projected downward, and those formed on the lower part of the retina are projected upward.

The eyeballs are separated laterally, on the average, 64 mm. in adult eyes. In looking at a distant object, if the axes of the eye are parallel, the images are formed on corresponding points of the retinas, but when the object is at some nearer point, the eyes must be turned inward in fixating the object, to compensate for their lateral separation. This function of the eyes is termed *convergence.*

The eyeball is rotated inward by the internal rectus muscle, so that its visual line is directed toward the object. This function is very closely associated with that of *accommodation;* one cannot act in any very great degree without the other also coming into play. The movement inward of the eye is measured by the angular deviation of the visual line, termed the *angle of convergence.*

The unit of convergence is the angle through which the visual axis moved to fixate on a point 1 meter distant. This is termed *1-meter angle of convergence* (Nagel; Fig. 25). If the object fixated is only $\frac{1}{2}$ meter distant, the movement will be twice as great; it is then 2-meter

angles. A point at $\frac{1}{3}$ meter would require 3-meter angles, and so on; 10-meter angles of convergence mean that the eye is directed to a point only $\frac{1}{10}$ meter distant.



FIG. 25.—Meter angles of convergence (Landolt).

Meter Angle.—In the figure, *O* and *O'* represent the centers of rotation of the two eyes; *O–O'* is the distance between these points, termed the *interocular distance*. It is measured by the distance between the pupils during fixation for remote objects. *O–M* is one-half this distance.

The line *C–M* is perpendicular to *O–O'*. When the object is situated on the line *C–M*, the convergence of each eye is equal. When the visual lines *J–O* and *J'–O'* are parallel, the angle of convergence is *nil;* when, however, the visual lines are directed to *C'*, 1 meter distant, *O–J* has deviated to *O–C'*. *J–O–C'* is the angle through which the visual line has moved to fix on *C'*. This is 1-meter angle of convergence.

C–M being parallel to *J–O*, *O–C'–M* is equal to *J–O–C'*.

In the right-angled triangle O–C'–M, O–M equals one-half the interocular distance.

O–C' = the distance of the point of fixation.

$\frac{O\text{–}M}{O\text{–}C'}$ = the sine of the angle O–C'–M.

The average interocular distance is 64 mm. O–M = $\frac{1}{2}$ of 64, or 32 mm. O–C' is 1 meter distant.

$\frac{O\text{–}M}{O\text{–}C'} = \frac{32}{1000} = .032$ = the sine of 1-meter angle. This corresponds to 1° 50′.

If the eye is directed to a point $\frac{1}{2}$ meter distant, C'', the visual line will deviate twice as much—that is, it deviates 32 mm. at $\frac{1}{2}$ meter distance. If the point of fixation is only $\frac{1}{10}$ meter distant, the amount of convergence will equal 10-meter angles.

To find the value of this in degrees we employ the same formula as above:

$\frac{O\text{–}M}{O\text{–}C^{10}}$ = sine of angle O–C^{10}–M. O–M = 32. O–C^{10} = $\frac{1}{10}$ meter = 100 mm.

$\frac{32}{100}$ = 0.32, the sine of angle of convergence = 18° 40′.

The value of the meter angles in degrees is obtained very nearly by multiplying 1° 50′ by the number of meter angles. The value of the meter angle varies with the interocular distance, and as there is considerable difference in this distance, a separate calculation is necessary for each individual.

A more simple method of determining the value of the meter angle is to find its relation to the centrad. The centrad is a prism which deviates a ray the $\frac{1}{100}$ part of the radius, measured on the arc (see page 19). The deviation of the meter angle is measured on the sine. For the angles obtained, the sine and arc are almost equal.

One-meter angle equals a deviation of 32 mm. (the average distance between the centers of rotation of the eyes being 64 mm.) at 1 meter distance = 32 in 1000 mm., or 3.2 in 100 = 3.2 centrads. One centrad = 0.57295°; 3.2 centrads = 1° 50′. Ten-meter angles equal a deviation of 32 mm. in $\frac{1}{10}$ meter, 100 mm., 32 in 100, or 32 centrads = 18° 20′. A 32-centrad prism not only gives us the value of 10-meter angles of convergence, but, placed before the eye with the base inward, it takes the place of 10-meter angles of convergence, so that the eye, without any convergence, would see an object on the line C'–M 10 cm. distant, as if it were situated at a remote distance.

The convergence becomes *greater* as the point of fixation approaches *nearer*. The number of meter angles is, therefore, inversely proportional to the distance expressed in meters. We thus designate the convergence in terms which indicate the same number of units of convergence as the diopters of accommodation necessary for the same distance. An emmetrope, in looking at an object 0.25 meter distant, would employ 4-meter angles of convergence and 4 diopters of accommodation.

The **amplitude of convergence** is the number of meter angles of convergence which the eyes can call into action. It is measured from the *far point* of *convergence* to the *near point* of *convergence*.

The far point of convergence is the point to which the visual lines are directed when the convergence is relaxed to its utmost; the near point of convergence is the point to which the visual lines are directed

when the convergence is at its maximum. If in the minimum degree of convergence the visual lines are parallel, the far point of convergence will be at an infinite distance. Usually the visual lines actually diverge forward at the minimum of convergence, constituting an outward squint, and converge by their posterior extremities toward a point behind the eyes. When this is the case, the far point and a portion of the amplitude of convergence are negative. In some cases, with the convergence relaxed to its fullest extent, the visual lines still deviate inward, constituting an internal squint. The convergence in such a case will be entirely positive.

Relation Between Accommodation and Convergence: Relative Accommodation.—While the two functions of convergence and accommodation, as has been previously explained, are closely associated, there is still some independence of action. In other words, it is possible to accommodate several diopters without convergence and to converge several meter-angles without accommodation. If the visual axes converge to a given point, the accommodation may be increased to a certain limit. The increased amount of accommodation exercised in these circumstances is measured by the ability to overcome concave glasses while the object still remains distinctly in view, and is denominated *the positive part of the relative accommodation.* It is also possible, while the visual lines converge for a given near point, to relax the accommodation from its association with that degree of convergence by placing convex glasses before the eyes, the object still remaining distinctly in view. This relatively diminished amount of accommodation is called *the negative part of the relative accommodation.* That convergence may be altered while the same effort of accommodation is maintained is demonstrable by placing a prism with its base inward before one eye, which then rotates outward, in order that the object may be seen singly, this object at the same time being perfectly distinct. Evidently the same effort of accommodation has been maintained, although the convergence of the visual axes is altered. At the far point of accommodation and convergence the accommodation has somewhat more play; at the near point, however, convergence has much the larger movement. The amplitude of convergence does not always diminish with age, as does the accommodation. Some persons, however, have a diminished convergence power or endurance, owing to changes in the ocular muscles similar in kind, though less in degree, to the senile changes which usually occur in other parts of the muscular system. Lucien Howe has designed an apparatus for the clinical measurement of the relative accommodation at the near point.

CHAPTER II

EXAMINATION OF THE PATIENT AND EXTERNAL EXAMINATION OF THE EYE; FUNCTIONAL TESTING

A systematic examination of each patient should be made in order to secure the preservation of careful records. For this purpose the following order of examination may be used:

Name and residence.

Age, sex, race, married, single, or widowed.

Family history: hereditary tendencies; general and ocular health of parents, brothers, sisters, etc.

Personal history: children, their general and ocular health; pregnancies, miscarriages; menopause; former illnesses; syphilis, gonorrhea; tuberculosis; injuries.

Occupation: relation of work to present indisposition.

Habits: brain use; tobacco; alcohol; tea and coffee; narcotics; sexual.

Date and mode of onset and supposed cause of present trouble; outline of its course.

Organs of digestion: teeth; mouth; tongue; tonsils; stomach; intestines.

Organs of respiration: nose, accessory sinuses; throat; lungs.

Organs of circulation: heart; pulse; blood.

Kidneys: examination of urine.

Abdominal organs: liver; spleen.

Organs of generation; menses; leukorrhea; uterine disease; urethral and seminal-vesicle and prostatic disease.

The skin, previous and present diseases. Endocrine organs.

Wassermann test of the blood and spinal fluid and tests for tuberculosis: fixation-complement test for gonorrhea.

Metabolic analysis.

Nervous system; intelligence; evidences of hysteria and psychasthenia; hallucinations; sleep; vertigo; gait; station; tendon- and muscle-jerks; paralysis, tremor; pain; subjective sensations; convulsions; headaches and their position.

Eyes: previous attacks of inflammation; injuries; infections; ocular palsy or squint; amblyopia; previous use of glasses; ability to use eyes.

Direct inspection and examination of eyes: inspection of the skull and orbits (symmetry or asymmetry); ciliary borders; puncta lacrimalia; upper and lower culdesacs; conjunctivæ; blood-vessels of the conjunctiva and episclera; caruncles; corneæ (oblique illumination and loupe; corneal microscope and Gullstrand slit lamp); irides (mobility and color); anterior chambers (depth and character of contents); vision; accommodation; balance exterior eye muscles; prism-convergence; prism-divergence; sursumvergence; convergence near point; position of eyes; mobility of globe; tonometer; light-sense; color-sense; fields of vision; field of fixation; ophthalmoscope; slit-lamp ophthalmoscopy, red-free light ophthalmoscopy; opthalmometer; retinoscope; test-lenses.

This schedule of examination must be modified to suit individual cases, as the patient presents trivial local lesions directly discoverable by inspection, or forms of disease requiring detailed study for their proper interpretation.

Direct Inspection of the Eye.—After the preliminary examination which the patient demands, the surgeon proceeds to the direct

inspection of the eye. The surface of the lids should be examined for swollen superficial veins, a common index of inflammation of the globe; their edges for inflammation, parasites, and misplaced cilia; the puncta for permeability, pressure at the same time being made over the lacrimal sac in order to express from it through the puncta any contained fluid; the upper and the lower conjunctival culdesac for accumulated secretion, granulations, and foreign bodies; the palpebral conjunctiva for hardened secretion in glands; the caruncles for swelling, attached foreign bodies, and irritation by incurved cilia; and the conjunctiva for the information to be derived from its blood-vessels.

In order to evert the lid, observe the following rule: Require the patient to turn the eye strongly downward, seize gently the central eyelashes of the upper lid between the index-finger and thumb of the left hand, draw the lid downward and away from the ball, place the point of the thumb of the right hand above the tarsal cartilage of the lid which is to be everted, the remaining fingers being steadied on the brow,

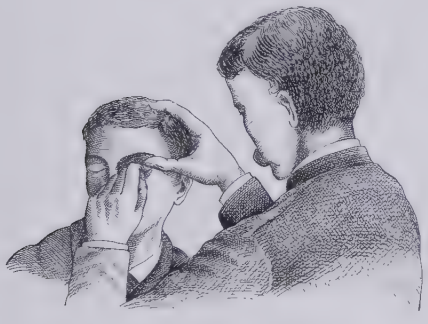


FIG. 26.—Position of hands in the act of everting the eyelid.

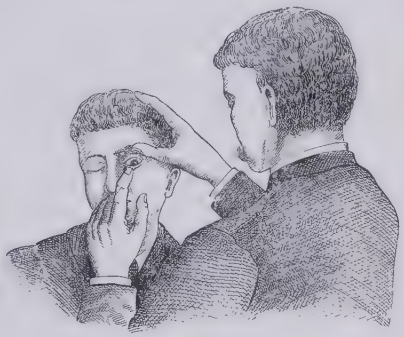


FIG. 27.—Eyelid everted for examination of its under surface and the upper part of globe.

and, by a quick movement, turn the edge of the lid over the point of the thumb, while this is simultaneously depressed. During the entire maneuver insist upon the downward direction of the patient's eyes; otherwise the lid cannot be turned without undue force and pain. If there are no lashes on the upper ciliary margin, the lower lid should be pushed beneath the edge of the upper in such a manner that it acts as a wedge on which the superior lid is everted. In this manner the lid can be everted with the fingers of one hand. Care should be exercised to expose the tissues of the upper culdesac which lie beneath the folded lid by making a second eversion of this folded lid, or by pushing the concealed fold into view by means of a blunt probe.

The surgeon should inspect the skin of the face and forehead, examine the orbits by palpation, ascertain the action of the orbicularis by causing the patient to close his eyes as if in sleep, and study the length, width, and symmetry of the palpebral fissures and the condition of the commissural angles.

Blood-vessels of the Conjunctiva.—In health only a few conspicuous blood-vessels are evident; in inflammation many more become visible. The arteries of the conjunctiva are derived from the palpebral and lacrimal branches of the ophthalmic; those of the episcleral tissue arise from the anterior ciliary branches of the ophthalmic, while the border of the cornea is surrounded by a plexus of capillary loops derived from the anterior ciliary vessels. This blood-supply may be conveniently divided, according to the late Mr. Nettleship, into three systems:

System I.—Posterior conjunctival vessels, whose congestion produces a bright red, velvety color, moving, on pressure of the eyelids, with the shifting of the conjunctiva, usually associated with mucopurulent secretion, and indicating conjunctivitis. Conjunctival congestion is most intense at the fornix and in its neighborhood, and decreases as the corneal margin is approached.

System II.—Anterior ciliary vessels, composed of perforating and non-perforating arteries and veins. The perforating arteries, which supply the sclera, iris, and ciliary bodies, are the branches seen in health entering about 5 mm. from the corneal margin, their points of entrance, in dark-complexioned people, often being distinctly tinted.

The non-perforating (episcleral) branches, invisible in the normal eye, produce, if congested, a pink zone surrounding the cornea ("ciliary congestion," "circumcorneal zone"), not moving on pressure of the lids with the shifting of the conjunctiva, unassociated with purulent discharge, which is one of the indications of iritis. Ciliary congestion is most distinct around the corneal margin and lessens as the fornix is approached.

The perforating veins and their non-perforating (episcleral) twigs, when congested, create a zone of dusky hue, often a symptom of glaucoma, or appear in unequal, deep-seated patches of lilac or violaceous color, indicating cyclitis or scleritis.

System III.—Anterior conjunctival vessels and the plexus of capillaries surrounding the cornea, derived from anterior ciliary vessels through whose numerous small branches anastomosis between System I and II takes place. Their congestion produces a circle of bright red injection, often partly on the cornea, a sign of inflammation of this structure, and typified in the early vascular stages of interstitial keratitis (see page 298).

In addition to these three varieties of congestion, numerous departures are noticeable, making it impossible to separate the form and specify the individual system involved. There may be a definite local injection, for instance, leash of vessels passing to a corneal ulcer; or all the systems are commingled in a general inflammation.

The blood-vessels of the conjunctiva can be well observed with a Zeiss corneal microscope, especially with the aid of a slit- or diaphragm-lamp. Luedde and Dennis by this means have made valuable observations in their studies of the conjunctival circulation in its relation to the early signs of arteriosclerosis. According to Dennis the changes

consist in a clogging of the corpuscles in the lumen of the vessels; emptiness of the vessel in front of the obstruction; beaded formation in the lumen of fine vessels; in advanced cases complete occlusion of vessels.

Temperature of the Conjunctival Sac.—According to Silex, the temperature of the lower human conjunctival fold is 35.55° C. (95.99° F.)—*i. e.*, about 2° C. lower than that of the rectum. There is an average increase of 0.98° C. in inflamed eyes, the highest temperature being found in acute iritis. The temperature of the cornea is about 29° C. or 84.2° F. (Leber).

Inspection and examination of the cornea reveals inflammation, vascularization, ulceration, opacities, and foreign bodies. Slight irregularities are detected by placing the patient before a window, while the eyes are made to follow the uplifted finger held about 1 foot from the face, and moved in various directions. The image of the window-bars reflected from the cornea will be broken as it crosses the spot of inequality.

A more accurate method is to employ a *keratoscope* (Placido's disk). This instrument consists of a disk shaped like a target, upon which are drawn concentric black circles, upon a white ground, a sight-hole being in the center. The patient is placed with his back to the window, while the surgeon holds the instrument in front of the eye, and, looking through the central aperture, observes the reflections of the circles from the cornea. If these are broken or distorted, the indications of irregularity in the surface are present. With this instrument corneal astigmatism (regular or irregular) may be detected. If present in considerable degree the reflections of the rings cease to be perfectly circular; they assume the character of an ellipse or exhibit irregular bulgings.

The cornea may also be examined with aid of *reflex images*. The patient is brought into relation with a candle flame placed at the side of his eye. With the aid of a loupe the *posterior* corneal reflex can be seen, and if, while the candle is shifted, and this reflex and the *anterior* reflex approach each other, any abrupt change in the distance between them takes place, it indicates a change in one or other of the corneal surfaces. This method is also useful in detecting imperfections in the evenness and polish of the cornea according as the images become irregular or lose their distinct outline. The cornea may also be investigated with the ophthalmoscope (page 106). (See also page 311.)

Staining of the Cornea.—Minute abrasions and ulcers may be found by dropping on the eye a concentrated alkaline solution of *fluorescein* (Gruebler's fluorescein, 2 per cent.; carbonate of soda, 3.5 per cent.), which colors green that portion of the cornea deprived of its epithelium, or in which the corneal epithelium is diseased, while the healthy epithelium remains unaffected. Epithelium in the immediate neighborhood of a corneal ulcer, although apparently not involved in the process, will also take the stain, as pointed out by Benson. Cocain solution instilled prior to or after the application of fluorescein distinctly enhances its staining properties, and the epithelium of the cornea, which has been softened by repeated instillations of cocain, will take on the

fluorescein stain. Where the lesion is not very recent, or where it is covered with necrotic tissue, the coloration will be yellowish or yellowish green. This substance also reveals defects of the endothelium of the cornea, and E. von Hippel maintains that it produces a deep-seated coloration of the cornea only when the endothelium is absent or diseased (see page 373). But it does not stain Descemet's membrane, and if the ulcer is deep only its sides accept the stain. C. A. Wood prefers a 2 per cent. solution of potassic fluorescide without the preliminary use of cocain. Toluidin-blue, as suggested by Veasey, and methylene-blue and eosin may likewise be used as coloring agents.

The Width of the Cornea.—This may be measured approximately by holding before it a rule marked in millimeters, and noting the number of spaces its width occupies, or with Priestley Smith's *keratometer*, which consists of a scale situated between two planoconvex lenses. The average horizontal diameter of the normal cornea is 11.6 mm. (Priestley Smith).

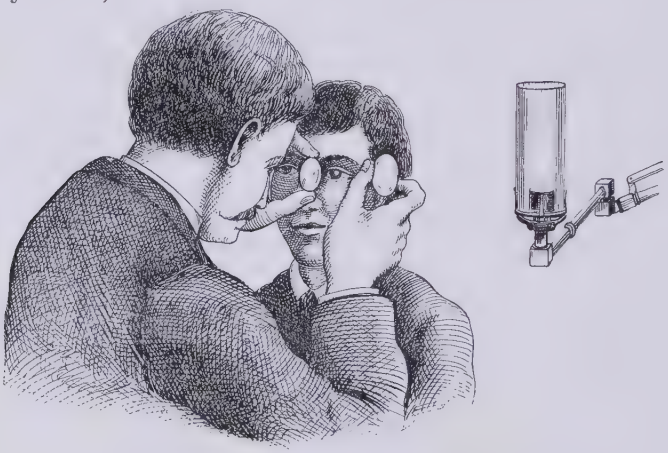


FIG. 28.—Method of oblique illumination.

The Sensibility of the Cornea.—This may be tested by gently touching the surface of this membrane with a wisp of cotton twisted to a fine point. If sensation is normal, the touch should be instantly followed by the reflex act of winking (*palpebral reflex*), although even if the cornea is insensitive closure of the lid may occur when the test-object comes into the field of the pupil. This is not due to contact, but represents the *retinal lid-closure reflex*. In organic anesthesia the *lacrimal reflex* is wanting, but is present in hysteric anesthesia. The opposite eye should always be tested as a control.

Oblique illumination is a method of examination by which the cornea, the anterior chamber, the iris, and, if the pupil is dilated, the lens and even the anterior layers of the vitreous may be studied. The surgeon places the patient 2 feet from the source of illumination, and focuses a beam of light with a 2- or 3-inch lens upon the cornea, at the same time observing the surface under examination through a lens of

the same focal distance, held between the thumb and forefinger, the disengaged fingers being utilized to elevate the upper lid (Fig. 28).

The distance of the lens must be varied slightly, according as the cornea, iris, or crystalline lens is brought within its focus, the patient being required to look up, down, and to each side, while all the anterior surfaces and media of the eye are illuminated. In order to detect foreign bodies in the cornea, the light should be directed at an acute angle. If the posterior pole of the lens is to be examined, the light is thrown perpendicularly into the pupil, the surgeon placing his eye in the same direction without interfering with the light.

By this method minute abrasions, previously undetected foreign bodies, channels of old vessels, and other corneal changes may be examined. The character of the aqueous humor, the depth of the anterior chamber, the surface of the iris, the presence of synechiæ, small tumors, atrophic fibers, and persisting pupillary membrane are evident, and, finally, opacities in the anterior capsule and axis of the lens are discoverable.

The use of oblique illumination by focusing daylight on the cornea with a lens and examining its surface from above with a strong magnifying glass, as Duane maintains, affords distinct advantages not only in detecting small lesions but also distortions of the cornea reflex (compare page 51, Inspection of Cornea).

The Corneal Loupe.—This is a lens, properly mounted, by means of which the cornea is strongly magnified, and which should be employed with oblique or electric flash-lamp illumination.

Dr. Edward Jackson has designed a *binocular magnifying lens* which possesses material advantages. Berger has constructed on the same principle a useful binocular corneal loupe, the value of which is enhanced by the attachment of an electric lamp as designed by Shumway, and E. Treacher Collins has arranged a binocular magnifier mounted on a spectacle frame, similar to the Hess loupe, which also carries an electric lamp. The *Zeiss binocular magnifier* is a most useful instrument, its optical system being so constructed that plane surfaces appear flat to the eyes.

Corneal Microscope.—A "corneal microscope" or a specially prepared lens of high power permits the study of minute changes in the cornea and in the iris, and is utilized for the examination of the traces of former vascularization, particularly after interstitial keratitis (see page 297), and by its help even the circulation of the blood in the vessels of a pannus may be studied. The Zeiss corneal microscope is particularly valuable in these respects, and with the aid of proper illumination (slit lamp) furnishes a method of investigating all the structures of the anterior portion of the eyeball of the most satisfying character.

The Diaphragm Lamp.—The slit lamp, being Gullstrand's modification of the Nernst lamp, is a special type of the *diaphragm lamp.* Should the diaphragm contain a circular aperture, another type results, for which Gullstrand proposes the name *hole lamp.*

Such lamps are essentially instruments whereby the observer is able to project a narrow beam or line of intense light upon the part of the eye to be examined. Quoting Gullstrand's words, "The source of light must be uniform and sharply limited, with a high specific intensity, and a sharp optical image of this source of light must be procured, that is, this light is condensed by an aplanatic lens into a sharp image which is thrown on the limited area it is desired to investigate, which is then studied with a corneal microscope." Because the illumination is intense a high magnification may be utilized. Various methods of using the light are employed, for instance, by direct lateral illumination, by transillumination, and by examination of reflecting surfaces in limited intensely illuminated areas.

Thanks to Gullstrand's researches and discoveries with the slit lamp and its various devices and sources of illumination, minute studies of the conjunctiva, cornea, iris, lens and vitreous are possible, far exceeding in value those secured by ordinary lamps and lenses; in short, a practically new field of investigation in ophthalmology has been developed, to wit, the *microscopy of the living eye*. In this respect we are especially indebted to the work of A. Vogt and of Koeppe. In this country the method has received particular attention from R. von der Heyt, Harry Gradle, and A. J. Bedell. Certain clinical applications of the slit lamp will be found in association with the description of some of the diseases and lesions of the various ocular tissues which are suited to this type of examination.[1]

Contact Illumination.—Basil Graves has developed a method of examination of the anterior segment of the eye, especially the cornea, by contact illumination, which he secures with a tube which ends in a plate of glass, neatly fitted and smoothed so that it can be pressed directly against the cornea and conjunctiva. Within this tube a second one slides, carrying in its end an electric lamp. A lamp similar to the one used in electric ophthalmoscopes furnishes the light. The lamp is placed in contact with the sclera, with only its edges overlapping the cornea, and thus brings out slight opacities. According as the position of the illuminator is shifted, a good light is obtained for the examination of other parts of the anterior segment of the eye, with the aid of a magnifier.

[1] Naturally, in a text-book it is not possible to describe this method in detail. For the convenience of the reader the following references are appended:

1. Atlas of Slit Lamp Microscopy of the Living Eye, by Alfred Vogt. Authorized translation by Robert Von der Heyt, Chicago, Berlin: Julius Springer, 1921.
2. Die Mikroskopie des lebendes Auges, von Dr. L. Koeppe, Berlin: Julius Springer, 1920.
3. Étude microscopique de l'Oeil Vivant, par E. Gallemaerts et G. Kleefeld, Paris: Octave Dom, 1920.
4. On Diaphragm Lamps in Ophthalmology, by A. Gullstrand, Trans. International Congress, Washington, 1922.
5. The Use of the Gullstrand Slit Lamp, by Harry Gradle, Arch. of Ophth., 1922, li, 264.
6. Observations on the Iris in Health and Disease as seen with the Slit Lamp, by Arthur J. Bedell, Trans. of Section of Ophth. of the A. M. A., 1922; Ibid., 1923.

The Color of the Iris.—The color of the irides varies: blue and gray are the predominating hues in northern countries; brown occurs next in frequency; while the various admixtures produce yellow and green shades. Black irides are never seen; but dark irides, taking into account the whole population of the world, are of the most frequent occurrence. The color of the iris depends upon the amount and location of the pigment in it. Thus, if the coloring-matter does not exist in the stroma, but only in the posterior layer, the blue iris is evident, but if there is much pigment in the stroma, the brown or dark brown iris appears. The color of the iris of practically all newborn children, negro infants not excepted, is of a light, grayish blue; the stromal pigment is developed subsequently.[1] According to Schindler the irides of badly nourished very young children, especially those with disturbances of digestion or metabolism, may change to a gray or brownish tint, at an age when as a rule normal irides still retain the blue color.

Slight differences in shade between the two irides are not uncommon; more rarely, even in health, the irides differ in color (chromatic asymmetry, *heterochromia iridis*), one being brown or greenish, the other blue or gray. Almost invariably, in cases of this sort, one iris corresponds in color with the irides of one parent, and the remaining iris with those of the other parent. Instead of uniform pigmentation, a single triangular patch or several irregular spots of dark color may appear upon one or both irides (piebald irides). Such dark spots, if small, have been mistaken for foreign bodies by inaccurate observers. This condition is sometimes temporary. Chromatic asymmetry, while perfectly compatible with health, has been observed in patients with neuropathic tendencies—chorea and epilepsy (Féré); the pupil of the blue eye may be smaller than that of the fellow eye; physiologic albuminuria may be present (T. Harrison Butler).

In many instances there is liability to disease on the part of the lighter eye (cataract, cyclitis, glaucoma); indeed, the evidences of cyclitis, according to Fuchs, are nearly always present. For this complicated type T. Harrison Butler suggests the name *heterochromic cyclitis*. Calhoun prefers Fuchs' descriptive term—*chronic cyclitis with decoloration of the iris*. Examination reveals punctate keratitis, vitreous opacities, sometimes choroiditis and cataract; glaucoma may develop. The opaque lens may be extracted and the results of operation are usually successful (Butler, Ellett, Knapp). A. Knapp has noted reaction to subcutaneous tuberculin tests and improvement under the influence of tuberculin therapy. Heterochromia iridis may appear in several members of the same family. The deficiency of pigment has been attributed to a lesion of the cervical sympathetic; Calhoun believes that in a large percentage of the cases paralysis of the sympathetic is the responsible cause for this heterochromia through its trophic disturbances.

Discoloration from disease results in one iris being green, that of the

[1] Ely records two dark irides in more than 1000 newborn children; one child was a negro.

fellow eye being blue, and indicates iritis or cyclitis; it is often an early symptom of inflammation of the iris, and should be looked for in every inflamed eye.

The Pupil.—The size of the pupil in health varies with exposure to light and with accommodation and convergence. Changes in its width also depend upon the quantity of blood in the vessels of the iris, the elasticity of the iris-tissue, and certain mechanical conditions.

Under normal conditions the pupils—subject as they are to many influences—manifest certain fluctuations, amounting, according to Schwarz, to 0.3 mm., even where the chief factors are practically constant. The pupil is generally small in old age, in the newborn, and in eyes with hyperopic refraction; it is larger in youth, and in eyes with myopic refraction. Women are apt to have wider pupils than men. Exceptions to these statements are not infrequent, especially in so far as the relation of errors of refraction to pupil-width is concerned. Usually it is stated that the pupil is smaller in blue irides than in dark ones. Some recent investigations indicate that this is not the case. With the accommodation at rest, the diameter of the pupil varies in daylight from 2.44 to 5.82 mm., the average diameter being 4.14 mm. (Woinow). The pupil as seen through the magnifying lens formed by the cornea is about one-ninth larger than the actual pupil (Duane). The position of the pupil is a little to the nasal side of the center of the cornea, and, under similiar illumination, the pupils should be round and of equal size (see also page 58). Slight inequality of the pupils is sometimes seen in healthy persons, and may be a congenital condition. Several instances of *cat-like pupils* have been observed in human eyes. In these circumstances the pupil in strong illumination takes on the form of a narrow elliptical slit (Greeff).

In addition to the factors already detailed which influence the size of the pupil, the *adaptation of the retina* to light must be taken into account, as Schirmer has shown. The pupil is exposed to clear daylight coming through a large window 1 meter distant, and the eye is permitted an adaptation of three minutes. Under such conditions a difference in width of 0.25 mm. has been determined. For the physiologic size of the pupil thus obtained Schwarz prefers the term *adapted width of the pupil.*

It is much to be regretted that the recorded variations in the diameter of the pupil are commonly imperfect, and that the loose statements, "pupils dilated," "pupils contracted," "pupils medium-sized," "pupils sluggish," have crept into many reports.

Measurement of the Pupil.—The pupil can be measured approximately by holding before it a rule, marked in millimeters, and noting the number of spaces its width occupies. The chief objection to this method is that the distance subtended on the rule is less than the diameter of the pupil, in proportion as the distance from the observer's eye is less to the rule than to the pupil (Jackson).

A great variety of instruments, known as *pupillometers*, have been devised for the accurate measurement of the width of the pupil. A

simple and serviceable device is an instrument which consists of a scale of circles held close to the observed eye, the scale being rotated until that circle which matches the pupil in size is reached (Fig. 29). Priestley Smith's *keratometer* (see page 52) may be used for the same purpose. Haab's pupillometer, which consists of a number of black disks, varying from 1.5 to 8 mm. in diameter, arranged in a perpendicular row, with which the pupil is compared, is a useful instrument. Care must be taken that the hand using these instruments does not cast a shadow on the examined eye. Such examinations suffice for ordinary clinical work. For more exact determinations the photographic method of pupillometry is employed. In order to measure quantitatively pupillary reaction C. von Hess devised a differential *pupilloscope*, which is an instrument of precision, and with it, according to Hans Barkan, who has described it and its clinical applications, anomalies of pupil reaction can be detected which are not discoverable by other methods.[1]

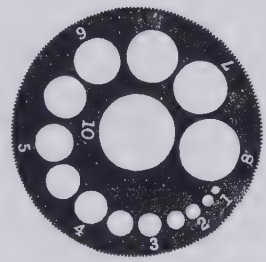


FIG. 29.—Simple pupillometer.

Mobility of the Iris—Pupil-reflexes.—The mobility of the iris is tested to find the presence of attachments between the iris and the lens (synechiæ), or atrophy of the iris, or to ascertain the sensitiveness to light of the retina or visual center.

Variations in the size of the pupil depend upon variations in the contractility of the iris and upon alterations in the lumen of its blood-vessels. These pupillary movements[2] are controlled by the *pupil muscles* (see page 60) which are set in motion either by reflex stimuli, or by association with other voluntary or involuntary movements, that is, by *synkinesis*, to adopt Parsons' term. Such movements are often called *pupil-reactions* or *pupil-reflexes*. They are as follows:

1. The *direct light-reflex of the pupil*—that is, the contraction of the pupil obtained by illuminating the pupillary area. It may be tested as follows: The patient is placed before a window in diffuse daylight, and one eye is carefully excluded. He is directed to look into the distance with the exposed eye, which is then shaded, and, if it is normal, a considerable dilatation of the pupil will occur. On removing the covering hand or card, contraction to the same size as that which existed before the test was applied takes place. The test may also be conducted in the following manner: The patient is seated as before described, and *both* eyes, which gaze steadily in the direction of the light, are covered by examiner's hand or a card, and after a few seconds the cover is removed from one eye and the initial width and the rapidity and completeness of the contraction of the exposed pupil are observed. The same procedure is repeated with the other eye. Again, inasmuch

[1] Arch., of Ophth, 1922, li, 29.

[2] This term is so well established and so commonly employed by clinicians that, in spite of the objections to it which have been urged, it should be retained.

as a properly lighted window is not always available, the test should be made with artificial illumination. The patient is seated in a dark room in front of the source of illumination (Argand burner, Welsbach light, lamp, or electric light) and looks into distance. Convergence and accommodation are relaxed, and the diameter of the pupil is measured with a pupillometer. Next, light is reflected into the eye with the ophthalmoscope mirror and the pupil reaction noted. Finally, the patient is required to face the light. The observer, standing on one side and watching the eye through a magnifying glass (Treacher Collins' binocular magnifier is excellent for this purpose), suddenly, by means of a lens, directs a beam of light directly on the center of the cornea. The slightest response can usually be detected. Satisfactory results are obtained if the source of illumination is a narrow beam of electric light obtained, for example, from a Würdemann transilluminator (see page 405) upon which a cap containing a small condensing lens is fitted, as in the model designed by Veasey. Very accurate observation can be made with the aid of the slit lamp; the light is turned off and on while the iris is studied with the corneal microscope. The faintest reaction can be seen (Feingold, Bedell).

2. The *consensual light-reflex*, or *indirect reflex action of the pupil*—that is, the contraction of the pupil of one eye, which is evident when the pupillary area of the opposite eye is illuminated. The test is made as follows: One eye is completely excluded from the source of illumination, and the other shaded in such a manner that the pupil can be observed beneath the cover. The completely excluded eye is next uncovered and the light directed into its pupil, the reaction which occurs in the shaded pupil being at the same time observed. Although the pupil of one eye acts under normal conditions in unison with its fellow, the direct and indirect reactions are not equal in intensity. According to Bach, the direct reaction to light is greater than the consensual. The statement, often made, that in normal eyes the pupils should be equal, not only with both eyes open but with one eye shaded, is not strictly correct, and usually the difference in width may be demonstrated by allowing for some seconds the stronger illumination to fall on one pupil (Bach).

3. The *accommodation- and convergence-reaction*, called also the *associated action of the pupil*, or the *accommodation synkinesis*—that is, the contraction of the pupil which takes place when the visual axes converge upon a near point. Usually the test is made as follows: The patient is required to look into distance and then quickly to direct his eyes at a near object—for example, the point of a pencil held at a distance of about 10 cm. Under normal conditions a contraction of the pupils will occur—that is, the sphincter of the iris contracts in association with the ciliary muscle and the internal recti. Bach's procedure is the following: The patient, seated facing a wall between two windows, is required to observe for twenty seconds a small white button placed 50 cm. from his eyes. The button is next gradually moved toward the eyes. No change in the pupil is observed until the object

reaches a distance of 40 cm. from the eyes. When a distance of 20–15 cm. is reached the contraction is stronger and may occur suddenly, associated with a strong convergence impulse. The amplitude of contraction, which is less marked than that which follows the action of light, varies between 0.25 and 0.75 mm.; exceptionally it is greater. Generally, it is less marked in old than in young persons. Refraction anomalies, according to Bach, produce no marked difference in the degree of contraction during convergence, except that in high myopia the reaction is sometimes delayed and less in amplitude. The associated movement of the pupil is much more closely connected with convergence than with accommodation; indeed, it is chiefly due to the impulse of convergence. If, experimentally, accommodation and convergence are dissociated, accommodation may take place without pupil contraction, but convergence cannot occur without contraction of the pupil (Swanzy and Werner).

4. The *sensory reflex of the pupil*, sometimes called the *skin-reflex*, or the *pain-reaction*—that is, a slight dilatation of the pupil which occurs on stimulating sensory nerves. It may be tested by pinching the skin of the neck, or, better, by applying to it a faradic brush.

5. The *cerebral cortex reflex of the pupil* is thus described by its discoverer, Dr. Haab: "If in a room illuminated only by a lamp or candle-flame, the light is placed so that it will shine laterally into a person's eyes while they look directly forward into the darkness, a marked contraction of both pupils takes place whenever the attention is directed toward the light, with no change in the position of the eyes. As long as the attention is directed to the light and fixation of the eyes on the dark wall is maintained the pupils remain contracted, but as soon as the attention is transferred to the point of fixation they dilate, although the quantity of light entering the eye has remained constant and all movements of accommodation and convergence are excluded."

The clinical significance of this reflex has not been ascertained, although Haab believes it may have some important bearing on the theory of attention, and that it should be investigated in all patients submitted to neurologic examination.

The observation of Piltz that in some persons the pupils contract or dilate when they call up a vivid mental picture of a bright or dark object has given rise to the term *imagination reflex of the pupils*.

6. The *palpebral (lid-closure) reflex of the pupil*, also denominated the orbicularis pupillary reaction, the Gifford-Galassi reflex, and the Westphal-Piltz reaction, was discovered by von Graefe. It consists in a contraction of the pupil which occurs when a forcible effort is made to close the lids. It has been explained by assuming that an associated stimulation of the sphincter nucleus takes place during closure of the lid; or it may be due to the mechanical effect produced by strong *contraction* of the orbicularis.

Should the stimulus be withdrawn after a pupil has contracted under the influence of light, convergence, or accommodation, it will return to the size it had before the stimulus was applied if the conditions remain

the same. This return or relaxation was called by Walter Jessop *the dilatation- or relaxation-reflex of the pupil.*

Innervation of the Iris and Explanation of the Pupil-reflexes.—The muscular tissue of the iris is divided into the *sphincter pupillæ*, a well-marked circular band of involuntary muscle surrounding the pupillary margin of the iris, and certain radially placed fibers, much less clearly marked, situated near the posterior surface, designated the *dilatator pupillæ.*[1] These two muscles are called the pupillary muscles, and each has a separate and independent motor-nerve supply which constitute the *miotic* and *mydriatic* nerves.

The third (oculomotor) nerve innervates the sphincter of the pupil, and contains the pupillo-constricting fibers which arise from its nucleus in the aqueduct of Sylvius. From this point the fibers proceed in the main trunk of the nerve to the orbit, and pass into the branch which supplies the inferior oblique, which they leave by way of the twig which constitutes the short root of the ciliary ganglion,[2] and finally arrive at the sphincter by the short ciliary nerves which penetrate the sclera around the optic nerve, and pass forward in the choroid and ciliary body to their destination in the iris. This line of communication between the nucleus of the third nerve and the sphincter of the pupil is called the *miotic* tract or *efferent* path, and is also known as the *centrifugal* pathway of the pupil reflex. Stimulation of it produces contraction of the pupil; section of it, moderate dilatation.

The cervical sympathetic innervates the dilatator of the pupil. The dilatator tract proceeds from a center in the medulla (or from a point in the aqueduct) into the lateral columns of the spinal cord as far as the third dorsal nerve. The pupillodilating fibers leave the cord by the ventral roots of the first, second, and third dorsal nerves and follow their communicating branches to the superior cervical ganglion. They pass upward in the ascending or carotid branch of the first cervical ganglion and arrive at the plexus around the internal carotid and the Gasserian ganglion. They reach the eyeball through the nasal branch of the ophthalmic nerve and its long ciliary nerves which perforate the sclera, and are distributed to the ciliary muscle and iris. The tract just described is called the *mydriatic* tract. Stimulation of it causes dilatation of the pupil; section of it, moderate contraction (Fig. 30).

Inasmuch as the iris is not under the control of the will, the contraction of the pupil which occurs when the eye is exposed to the source of light in the manner described is a reflex—that is, its motor nerves are excited to action indirectly by the reflex stimulus of light. This light reflex is under the control of the constrictor center, which the stimuli

[1] The existence of a dilatator muscle in the iris is denied by some authors, but the combined anatomic and physiologic evidence of its presence seems to be conclusive.

[2] As Langley and Anderson have shown, there is a cell-station in the ciliary ganglion. The root-fibers which belong to the oculomotor end in the ganglion, and with the cells of the ciliary ganglion a new neuron begins for the fibers which pass to the ciliary muscle and the sphincter of the pupil.

reach by passing along a tract which is known as the *afferent pathway*, the exact course of which is as yet uncertain, but it is probably somewhat as follows: The fibers of the pupil-reflex tract begin in the retina and arise from all parts of it and proceed in the optic nerves, and are in all probability histologically different from those which are concerned

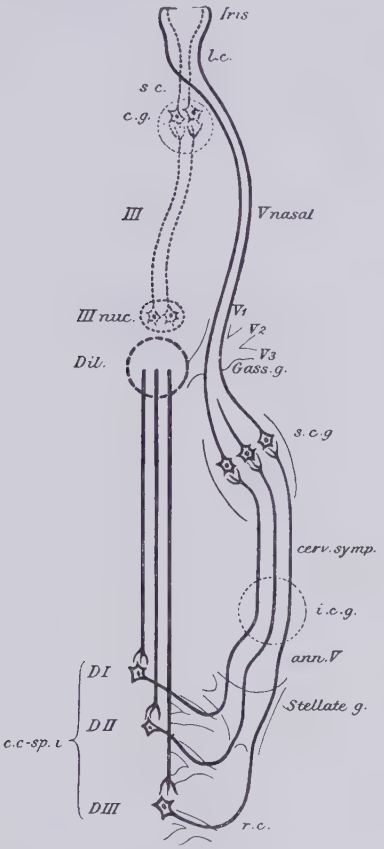


FIG. 30.—Diagram of the efferent pupillary paths. Dotted lines, pupilloconstrictor; *III nuc.*, nucleus of third nerve; *c. g.*, ciliary ganglion; *s. c.*, short ciliary nerves. Solid lines, pupillodilatator: *Dil.*, hypothetical dilatator center in the medulla; *c.c-sp. i*, Budge's centrum ciliospinale inferius; *DI*, *DII*, *DIII*, first, second, and third dorsal nerves; *r. c.*, ramus communicans; *Stellate g.*, stellate ganglion; *ann. V*, annulus of Vieussens; *i. c. g.*, inferior cervical ganglion; *cerv. symp.*, cervical sympathetic; *s. c. g.*, superior cervical ganglion; *Gass. g.*, Gasserian ganglion; *V1*, *V2*, *V3*, first, second, and third divisions of the fifth nerve; *V nasal*, nasal branch of the ophthalmic (first) division of the fifth nerve; *l. c.*, long ciliary nerves. (Description and diagram from J. Herbert Parsons.)

with vision.[1] In the chiasm these pupillary fibers undergo partial decussation and enter the optic tracts, which they leave just in advance

[1] According to some observers (Hess) the portion of the retina which receives the light rays, giving origin to the pupil-reflexes, is confined to a small central area with a radius of about 3 mm.

of the external (lateral) geniculate body, and reach the third nerve nucleus. From a special part of this nucleus, probably the small-celled median nuclei, the pupilloconstrictor fibers arise and reach the sphincter of the iris, constituting the efferent, miotic, or centrifugal pathway already described[1] (Fig. 31).

The direct light reflex of the pupil is the result of an active constrictor effect, the stimuli passing along the afferent pathway to the sphincter center in the third nucleus, and from there by the efferent pathway to the termination of the miotic fibers in the iris.

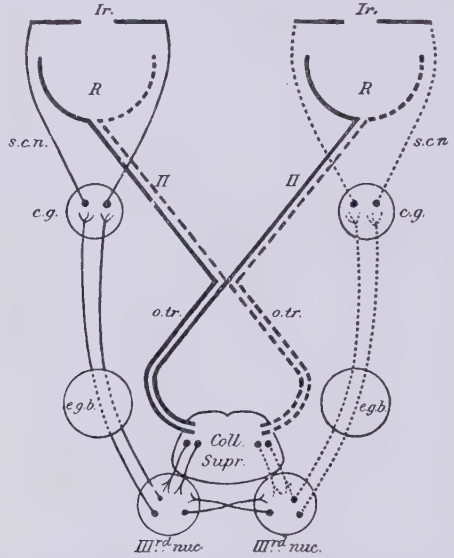


Fig. 31.—Diagram of the afferent and efferent pupillary paths for light stimuli. Afferent paths from left sides of retinæ, thick solid lines; afferent paths from right sides of retinæ, thick dotted lines; efferent paths of left eye, thin solid lines; efferent paths of right eye, thin dotted lines: *Ir.*, iris; *R*, retina; *II*, optic nerve; *o. tr.*, optic tract; *Coll. Supr.*, colliculus superior or anterior corpus quadrigeminum; *IIIrd. nuc.*, nucleus of third nerve; *e. g. b.*, external geniculate body; *c. g.*, ciliary ganglion; *s. c. n.*, short ciliary nerves. (Description and diagram from J. Herbert Parsons.)

The consensual or indirect light reflex of the pupil occurs because the stimulus passes to the opposite eye, either by reason of the decussa-

[1] The path by which the pupillary fibers leave the optic tract to reach the third nucleus, in the language of Parsons, is as yet conjectural. This author, referring to the pupillo-constrictor path, thinks it is probable that the fibers pass through the superior brachium of the quadrigeminal body to the superior colliculus, there making new connections with the cells which convey the impulses to the third nucleus of the same and also the opposite side. Von Hippel states that after the pupil fibers leave a tract in advance of the external geniculate body, they run up and in toward the median line, and as they enter the white substance of the corpora quadrigemina, they radiate, part of them going to the roof, and another part, under the aqueduct, toward the sphincter nucleus. It is probable that there is a connection between the two sphincter nuclei over the median line through the ganglion-cell processes.

tion of the fibers in the chiasm or because of its transference from one nucleus to the other.

The sensory reflex of the pupil is a dilatator reflex called into existence by various sensory stimuli. According to Parsons, it is due in part to augmentation of the dilatator tone through the sympathetic, and in part to inhibition of the constrictor tone.

The convergence and accommodation reaction of the pupil is not a reflex, but an associated movement, and has been ascribed to the effect of a stimulus which reaches the convergence center in the third nucleus, and is diffused to the cells which innervate the ciliary muscle. According to Schwarz, it is possible that a single cerebral impulse to accommodate both eyes to near vision stimulates simultaneously the nuclei which regulate convergence, accommodation, and pupil contraction.

The cerebral cortical reflex, or, better, reaction of the pupil, is of complex nature and results from psychic stimuli. The explanation of the lid-closure reflex of the pupil has been given (see page 59).

Not only may constriction of the iris, and therefore contraction of the pupil, be due to contraction of the constrictor (sphincter) muscle, but it also may be caused by relaxation of the dilatator muscle and dilatation of the blood-vessels of the iris. As before stated, the evidence strongly indicates that the light reflex is an active constrictor effect, although some writers maintain that it should be explained by an inhibitory dilatator influence. Instead of locating the center for the light reflex of the pupil in the small cells which occupy the median part of the third nucleus, Marina has placed it in the ciliary ganglion. Bach had described an inhibitory constrictor and an inhibitory dilatator center in the spinal end of the floor of the fourth ventricle, and, according to him, irritation of these centers will cause either dilatation or contraction of the pupil. The presence of these centers is denied by a number of observers.

Not only may dilatation of the pupil be due to contraction of the dilatator muscle (dilatator pupillæ), but may also be caused by relaxation of the constrictor (sphincter) muscle and by constriction of the blood-vessels in the iris. The dilatator pathway has been described (see page 60). Budge and Waller believed that the origin of the pupil-dilating fibers should be referred to the spinal cord in a region between the exits of the sixth cervical and fourth dorsal or thoracic nerve (probably opposite the seventh cervical and first thoracic), which is known as *Budge's ciliospinal center* (see Fig. 30). Although certain clinical and experimental evidence is in favor of this center, its existence has not been proved. Not only is the sympathetic pathway of the pupils concerned with maintaining a certain tone in the dilatator muscle of the iris, but it is also capable of being actively awakened by various sensory stimuli.

Dilatation of the pupil occurs in glaucoma, in optic-nerve atrophy, in orbital disease, and under the influence of mydriatics. It is further seen in fright, emotion, in deep inspiration or expiration, in anemia, in depressed nervous tone, aortic insufficiency, cutaneous

stimulation (*skin-reflex*), and irritation of the cervical sympathetic. If paralysis of accommodation is associated with dilatation of the pupil, objects may appear smaller than normal (*micropsia*).

In diseases of the nervous system, dilatation of the pupil, if of cerebral origin, indicates extensive lesion; if of spinal origin, irritation of the part. Systematic writers have divided dilatation into *irritation mydriasis*, caused by irritation of the pupil-dilating center of fibers, and *paralytic mydriasis* (*iridoplegia*), caused by paralysis of the pupil-contracting center or fibers.

In *irritation* or *spastic mydriasis* the pupil may be moderately or widely dilated. It reacts somewhat to light, accommodation, and convergence if the dilatation is not extreme; but if it is, these reactions may be lacking. Cocain usually produces no further dilatation of such a pupil, nor is it readily contracted by pilocarpin, and sometimes not at all. It is seen in hyperemia and irritation of the cervical part of the spinal cord, in spinal meningitis, in tumor of the cord, sometimes in tumor of the cerebrum, in acute mania, and in early tabes dorsalis, and paretic dementia. Spasmodic mydriasis has been observed in a healthy subject (Cramer).

In *paralytic mydriasis* (*sphincter paralysis*) the pupil is dilated, but not necessarily *ad maximum*. It does not react to light, accommodation and convergence, and the condition is sometimes described as *pupillary rigidity* or *total iridoplegia*. If there is only paresis and not paralysis of the sphincter, a sluggish reaction to light, accommodation, and convergence may be obtained. Cocain still further dilates a pupil of this character, and it is contracted by the action of pilocarpin.

Paralytic mydriasis may be caused by a lesion in the sphincter, the sphincter nucleus, or the centrifugal tract. It is seen in disease of the base of the brain affecting the third nerve or its nucleus, in cerebral lues, in pressure on the cerebrum great in degree, in late stages of meningitis, in edema of the cortex, in cerebral softening, and in hemorrhage of the centrum ovale and cerebral peduncles.

In *medicinal mydriasis—i. e.*, one caused by atropin or a similar drug or by certain toxins—there is also paralysis of accommodation.

Contraction of the pupil (*miosis*) appears in congestions of the iris, in traumatisms of the iris (*traumatic miosis*), in certain fevers, in plethora, venous obstruction, mitral disease, pulmonary congestion, paralysis of the sympathetic, and under the influence of miotics. During sleep the pupils are contracted. If spasm of accommodation is associated with contraction of the pupil, objects may appear larger than normal (*macropsia*).

If the miosis is of cerebral origin, it indicates an early irritative stage of the affection (meningitis, etc.); if of spinal origin, a depression, paralysis, or even destruction of the part.

Systematic writers have divided contraction of the pupil into *irritation miosis*, caused by irritation of the pupil-contracting center or fibers, and *paralytic miosis*, caused by paralysis of the pupil-dilating center or fiber. The same factors which cause miosis may cause myd-

riasis, the determining factor being the degree and the duration of the lesion.

In *irritation* or *spastic miosis* the pupil is contracted, in medium degree if one etiologic factor is active, *ad maximum* if both are concerned. Such a pupil dilates little or not at all in the dark, and usually is unaffected by the action of light. It is readily dilated by a mydriatic (atropin), and still further contracted by pilocarpin. The active lesion may reside in the iris, in the sphincter nucleus, or in the centrifugal pathway. According to some authors spastic miosis may be indirectly caused by failure of the inhibitory influences to act on the sphincter nucleus (Schwarz).

Irritation or spastic miosis may be caused by inflammatory affections of the base of the brain and the meninges in their early stages, by brain abscess, by beginning sinus disease. It is evident in the early period of cerebral neoplasms, in small hemorrhages in the cerebellum, at the onset of cerebral apoplexy, and in apoplexy of the pons. It is also seen in hysteria, at the beginning of epileptic attacks, in certain toxemias, in tobacco amblyopia, and under the influence of long-sustained efforts of accommodation.

In *paralytic miosis* (*dilatator paralysis*) the pupil is contracted, but its motility is preserved in that it reacts to light and the impulse of convergence. In the dark it dilates, but less perfectly than a normal pupil. Such a pupil is dilated by mydriatics, but only partially; it is contracted still further by miotics.

Paralytic miosis may be caused by lesions in the cord above the dorsal vertebra, and is especially noteworthy in tabes dorsalis (*spinal miosis*). It is also seen in paralysis of the insane, pseudodementia paralytica of syphilitic origin, in some forms of bulbar paralysis, and in some varieties of multiple neuritis (Mills). It is caused also by injury to the cervical sympathetic, or by pressure, for example, from enlarged cervical glands or an aneurysm. If the cervical sympathetic is paralyzed, with the miosis there are enophthalmos, unilateral anhidrosis, and ptosis (*sympathetic ptosis, Horner's syndrome*).

To a pupil which does not react, either directly or indirectly (consensually) to the influence of light, but contracts promptly on convergence of the visual axes, the term *reflex inactivity* or *immobility of the pupil* or *reflex iridoplegia* is applied. Usually, it is denominated the *Argyll-Robertson pupil*. Generally, the condition is bilateral, but unilateral reflex inactivity or immobility is also seen, and even where the failure of light reaction is bilateral, one pupil may be smaller than the other, although both are miotic pupils. Sometimes the same reflex immobility is present when the pupils are dilated. Frequently the affected pupils are not round, but slightly oval or pointed. The seat of the lesion in these circumstances is not certainly known. It has been placed in the fibers which pass from the proximal end of the optic nerve to the oculomotor nuclei by some authors, and by others is considered to be nuclear. Otto Barkan points out that with the pupilloscope (page 57) it is possible to detect a very early stage of an Argyll-Robertson

pupil. Following Hess, he states that somewhere in the region of the corpora quadrigemina the pupillary fibers proceed from the optic tract to the third nerve nucleus. These fibers are a link between the afferent and efferent limbs of the arc, and are designated as the "intercalated" or connecting portion of the arc. To a lesion of this path the Argyll-Robertson pupil is ascribed. Bach believes that it may be located in the spinal end of the sinus rhomboidalis, and Marina and Lafon place the causal lesion in the ciliary ganglion. John Dunn thinks that the Argyll-Robertson pupil is the result of the abolition of the autonomic reflex of the ciliary ganglia.

Reflex immobile or inactive pupils are especially noteworthy and frequent in tabes dorsalis (60–90 per cent. Uhthoff) and paretic dementia (50 per cent. Uhthoff), and, as is well known, may precede the general signs of these diseases by many years. They also occur in syphilis, especially cerebral syphilis (10 per cent. Uhthoff). Argyll-Robertson pupils probably can also be caused by hereditary syphilis, inasmuch as tabes has been noted in virgins with hereditary syphilis. Reflex iridoplegia has also been observed in non-syphilitic affections, in epidemic encephalitis, in disseminated sclerosis, in syringomyelia, in alcoholic neuritis, in methyl-alcohol poisoning with neuritis (A. Fuchs), and after injuries (Magitot). But in spite of these exceptions Argyll-Robertson pupils must almost always be suspected as an indication of a syphilitic or metasyphilitic affection and the usual serologic tests should be made. If miosis is present it has been attributed to a sympathetic affection and to tonic contraction of the sphincter, but Bach thinks that it depends upon an irritation of the reflex inhibitory center which he believes he has discovered. According to the same author the Argyll-Robertson pupil may remain unilateral for years, and exist as an isolated symptom.

The reverse of the Argyll-Robertson symptom has been observed, that is to say, the pupil reacts to light, but fails to react to convergence, and has been ascribed to disease in a special part of the oculomotor nucleus.

Unilateral reflex iridoplegia, or a condition in which one pupil is unaffected by varying degrees of illumination of both eyes, but reacts to accommodation, while the pupil of the other eye responds to a separate light stimulus of either eye, and which is seen in tabes dorsalis and syphilitic cases, should be distinguished from *unilateral reflex blindness*, caused, for example, by interruption of the conducting power of one optic nerve. In unilateral reflex blindness illumination of the pupil area on that side fails to elicit either the direct or the indirect pupil-reflex.

It is evident that in certain cases, as has been noted, although light perception is present, the reaction of the pupil to light is not demonstrable, for example, in changes in the iris itself, that is, inflammation, atrophy, laceration, etc., paralysis of the oculomotor nerve or its nucleus, and reflex iridoplegia. Occasionally, although light perception is absent, the reaction of the pupil to light is preserved. This may occur with lesions interrupting the optic pathway above the point where the fibers

of the reflex arc pass to the center for pupillary movements (Fuchs). Finally, in rare instances, although light perception is eliminated due to disease of the optic nerve, the reaction of the pupil is present. This phenomenon has been explained by assuming that the fibers concerned with the light reflex are more resistant than those which are concerned with visual impressions. The practical point is that response of the pupil to light stimulus does not necessarily prove the existence of light perception.

Convergence Anomalies of the Pupils.—As already noted, in complete rigidity of the pupil, such as occurs with total paralysis of the sphincter, there is no convergence reaction, but it is conceivable, as Schwarz points out, that a common disturbance of convergence reaction and light reaction, due to interruption of the corresponding pathways leading to the sphincter nucleus, may occur without paralysis of the sphincter.

Occasionally a pupil which is inactive to light stimulus, but which contracts on convergence, will remain in this contracted condition for a considerable length of time before it slowly returns to its original size. This phenomenon has been called the *myotonic pupil movement* by Sänger, and *neurotonic convergence reaction* by Piltz. It has been noted in tabes dorsalis, in multiple sclerosis, migraine, and alcoholism. Failure of the convergence reaction of the pupil, unassociated with disturbances of the light reflex, although rare, may occur. It may be complete or incomplete, and the convergence rigidity may be associated with paralysis of accommodation, or this may be absent.

The *palpebral reflex of the pupil*, according to Bach, may be seen in normal eyes, and is perhaps accountable for some of the contradictory observations which have been made on the pupil reflexes. If there is sphincter paralysis it is abolished, but sometimes it appears, although light reaction and convergence reaction are absent, when it must be assumed that the sphincter itself is not paralyzed, or, at least, not completely disabled.

Unequal Pupils (*Anisocoria*).—The statement that inequality of the pupils is always pathologic, considering the number of observations now on record, is subject to revision, and it would seem that we may safely speak of pathologic and non-pathologic anisocoria; in short, that slight differences in the width of pupils may be compatible with perfect ocular and general health. As Uhthoff points out in non-pathologic anisocoria the pupils are round and react normally to the ordinary stimuli, which usually is not the case in pathologic pupillary inequality. Tarun has well studied the subject in this country. If there is recent wide dilatation of one pupil and no disease of the eye, the instillation of a mydriatic may be suspected. Unequal pupils occur in eyes with widely dissimilar refraction, if one eye is blind, in aneurysm, pulmonary tuberculosis, dental disease, sinusitis, traumatism, and in diseases of the nervous system. If the disease is cerebral, the inequality denotes unilateral or focal brain disease. Anisocoria is not uncommon in tabes, disseminated sclerosis, and paretic dementia.

Varying inequality of the pupils (*springing* or *alternating mydriasis*) or a one-sided mydriasis, now occurring on the one side and now on the other, may be a premonitory symptom of insanity, and has been noted in general paralysis and locomotor ataxia. It is doubtful if it occurs in healthy persons, but the so-called false alternating mydriasis, according to Piltz and Frenkel, may be due to an inequality in the reflex excitability to light of the two eyes, or to inequality in the response to accommodation or spasm of the orbicularis muscles. Duane quotes Coats and von Hippel who have observed *cyclic contraction* and *dilatation* of the pupil, sometimes with dilatation and contraction of the palpebral fissure, in cases of oculomotor palsy.

Special and Paradoxic Pupillary Phenomena.—The *hemiopic pupillary inaction* is referred to on page 599. During anterior fixation of normal eyes *isocoria* (equality in the size of the pupils) is the rule. But it has been observed that during strongly maintained lateral fixation to the right or left *anisocoria* (inequality in the size of the pupils) takes place, the right or left pupil being the larger according to the circumstances. This is called *Tournay's reaction.* Dilatation of the pupil under the influence of light stimulus and contraction when it has been shaded have been described in cases of meningitis as *paradoxic pupillary reactions.* The phenomenon has been explained by assuming a reflex stimulation of the dilatator by a psychic influence, or that the action of the dilatator is indirectly increased because there is rapid exhaustion of the sphincter (Silex). *Paradoxic pupil dilatation* is the antithesis of the conditions just described, and has been observed frequently in experimental work in connection with the relation of the sympathetic to the eye. Paradoxic convergence reaction—that is, a dilatation of the pupil on convergence of the visual axes—has been recorded.

Hippus is a rhythmic contraction and dilatation of the pupil without alteration of illumination or fixation. It is a normal phenomenon due to the constantly varying sensitive and psychic reflexes, but it may occur in exaggerated degree in hysteria, mania, meningitis, and other diseases of the nervous mechanism, and has been observed in barbital poisoning (T. Fleischer) in the sense that a rapid variation of the pupils from miosis to mydriasis takes place.

Testing Acuteness of Vision.—The acuteness of direct vision is the power of distinguishing form and size, and is a function of the macula lutea. Vision with other parts of the retina, that is, indirect vision, is less distinct.

Visual acuteness, although it answers the requirements of a measure of the form-sense, that is, the recognition of the form of objects, is influenced by other visual factors, namely, the transparency of the ocular refractive media, the refraction of the eye and the condition of the retina and optic vision, and by the light-sense (page 70).

In order to determine the acuteness of sight, that is, to make the usual tests of vision, test-types are employed, in which the letters are of various sizes and constructed according to the methods described on page 35.

When it is desired to make the test, the patient is placed 6 meters from the type-card, which is properly illuminated, and each eye is tried separately. If the letters of No. 6 (20 feet approximately) are read, vision is normal, or 1, but if, at the same distance, letters no smaller than those numbered 18 (60 feet) can be discerned, vision is $\frac{1}{3}$. It is usual to express these results according to the formula $V = \frac{d}{D}$, in which V stands for visual acuteness, d for the distance of the patient from the card, and D for the distance at which the type should be read; so that in these instances the vision would be recorded $\frac{6}{6}$ and $\frac{6}{18}$, or in feet, $\frac{20}{\text{XX}}$ and $\frac{20}{\text{LX}}$. The rays coming from the letters at 6 meters' distance have so little divergence when they reach the eye that they are usually considered parallel. Hence, if the patient sees distinctly at this distance, his vision is perfect at the longest range (see also page 38). In point of fact, however, there is an appreciable divergence of the rays from the distance mentioned, equivalent to one-sixth of a diopter, and in the final adjustment of glasses this divergence should be recognized. Any other distance may be chosen, provided it does not place the patient closer to the test-card than 3 meters, at which close range the function of accommodation would introduce an element of inaccuracy. Thus, the scale of de Wecker assumes $\frac{5}{5}\left(\frac{15}{\text{XV}}\text{ approximately}\right)$ instead of $\frac{6}{6}$, as $\frac{1}{1}$. The *international test-types* are also designed for a distance of 5 meters and the visual acuteness is expressed in decimals. It is the custom of many ophthalmologists to record the visual acuteness in a decimal fraction, for instance, $V = 0.5$ instead of $\frac{6}{12}$. In like manner, a 4-meter distance may be utilized, as has been done by Edward Jackson. This author urges the standardizing of all cards of test-letters by comparison of the visibility of each line of letters with that of a graded series of broken rings which he has designed and has had prepared.

The acuteness of sight, as tested with types constructed on the basis of an angle of 5′, does not represent accurately the highest vision attainable, indeed, many good eyes possess a vision of $\frac{5}{4}$ of the standard angle. For this reason the late Dr. James Wallace arranged a series of test-types in which an angle of 4′ has been substituted as the basis of each letter.

For the purpose of a control test, and also for determining *visual acuteness of illiterate persons*, cards are employed on which a number of differently arranged dots are placed, of sizes which should be counted at different distances, and among these Burchardt's international tests are the most useful. For the same purpose incomplete squares corresponding in size to the test-letters have been constructed, the incomplete sides being turned successively in different directions or the broken rings proposed and designed by Landolt may be used. Wolffberg has

designed a useful test which consists of small pictures of well-known objects, which in size approximately conform to the standard angle. The whole subject of test objects for the illiterate has been admirably described by Dr. A. E. Ewing.[1]

If the patient fails to decipher the largest letters at the distance employed, he should be moved closer to the card; thus, he may be unable to read the type numbered 60 at 6 meters, but may discern this at 4 meters, $V = \frac{4}{60}$ or $\frac{1}{15}$ of normal. Still further depreciation of visual acuteness is recorded by requiring the subject to count the outstretched fingers at various distances, 0.2, 1, or 2 meters, V = counting fingers at 0.2 meter, etc. For determining the lower degrees of sharpness of vision by a method more precise than the one just described, Landolt's *optotypes* may be employed.[2] If the patient is unable to count fingers, his ability to perceive the movements of the hand at 0.5, 1, or 2 meters is tested, V = movement of the hand at 0.5 meter, etc. When the ability to distinguish form (*qualitative light perception*) no longer exists, the perception of light should be investigated by alternately screening and exposing the eye, or by illuminating the eye with light reflected from a mirror, or focused upon it with a condensing lens.

Binocular Visual Acuteness.—The visual acuteness of both eyes is usually better than that of each eye tested separately. This is a notable feature in correcting errors of refraction, in that the acuteness of vision is often distinctly improved if both eyes are concerned with the visual act, and this is true in the presence of anisometropia (page 160) and even where one eye is amblyopic.

Illumination and Acuteness of Vision.—Naturally, as has been stated, a properly illuminated type-card is essential for the accurate testing of visual acuteness. As satisfactory daylight illumination is often difficult to obtain, it is customary to utilize artificial light, and excellent cabinets containing the test-cards, and so constructed that the source of illumination (electric light) is evenly distributed over the surface of the card, are available. The illumination should not be below 3-foot candles (Parsons). It is inadvisable to use only one type-card; several of standard construction should be provided, but containing different sets of letters; otherwise the patient's memory may introduce inaccuracies into the records.

Light-sense.—Having determined the acuteness of vision by means of the test-letters, the examiner has ascertained the *form-sense*, and may proceed to try a second subdivision of the sense of sight, the *light-sense*, which is the power possessed by the retina, or center of vision, of appreciating variations in the intensity of the source of illumination.

An instrument called a *photometer* is employed for this purpose, and consists essentially of an apparatus—for example, the one designed by Izard and Chibret—by which the intensity of two sources of light may

[1] American Journal of Ophthalmology, 1920, vol. 3, No. 1, p. 5.

[2] Ophthalmic Record, 1899, vol. viii, p. 624.

be compared. The patient, looking into the instrument, sees two equally bright disks. One disk is now made darker, and the power of the eye to perceive the difference in the illumination of the two disks ascertained, or one disk is made entirely dark, and then gradually illuminated, and the smallest degree of light noted by which the patient can perceive the disk coming from the darkness. The former is called the *light-difference* (L. D.), and the latter the *light-minimum* (L. M.). In more exact language, to quote Percival Hay,[1] who has designed a photometer, by light-difference is meant the minimal difference capable of being perceived—the threshold of discrimination, and by light-minimum is meant the minimal stimulus capable of being perceived—the threshold of sensibility. By means of Förster's photometer the lowest limit of illumination with which an object is still visible (the *minimum stimulus*) is ascertained. For determining the "light-minimum" R. Wallace Henry's photometer is very useful. Ives and H. Maxwell Langdon have improved the accuracy of the last-mentioned instrument by using electricity with a rheostat and ammeter in the circuit as the source of illumination, and having the amount of light controlled by an iris diaphragm instead of varying thicknesses of opal glass, as suggested by Henry. Percival[2] has constructed some simple tests for determining the light-sense composed of celluloid disks which can be rotated; the L. M. disks are two black disks on each of which are three white sectors of differential sizes, and when one of these disks is rotated three lighter rings are seen of which the inner is the lightest; the L. D. are white disks with black sectors of correspondingly different sizes, and when rotated indicate the light-difference for various values. Some information in regard to the light-sense may be obtained by testing the acuteness of vision by means of two cards, under a different degree of illumination, and by comparing the results with a similar examination of a subject believed to have normal power of appreciating different degrees of illumination. De Wecker's *photometric types* may also be employed. These consist of white letters placed upon gray backgrounds of different intensities. The light-sense may also be tested with gray letters on a gray ground, those of Bjerrum being constructed on the same principle as Snellen's types.

Retinal Adaptation.—On passing from daylight into a darkened room at first practically none of the surrounding objects is visible; gradually, to use an ordinary expression, the eyes "become accustomed to the darkness" and are able to see more and more distinctly. The complete adjustment of the eyes to the surrounding illumination is known as *adaptation*, which takes place because the retinal purple, bleached by light, is regenerated by degrees. Adjustment of the eye for the dark is denominated dark-adaptation or *scotopia*, and for bright light, light-adaptation or *photopia*, using the terms employed by Parsons. In testing the light-sense it is necessary to allow a period of time

[1] Archives of Ophthalmology, 1905, vol. xxxiv, p. 160.

[2] Transactions of an International Congress of Ophthalmology, Washington, D. C., 1922.

for adaptation (ten minutes is sufficient in ordinary examinations) during which the patient remains with bandaged eyes in a dark room. In place of a photometer, for example, Förster's, an instrument known as an *adaptometer*, of which there are several models, may be advantageously employed.

Color-sense.—A third subdivision of the sense of sight is the *color-sense*, or the power which the retina has of perceiving color, or that sensation which results from the impression of light-waves having a certain refrangibility. This examination is of especial interest in the detection of *color-blindness* (see page 572).

1. **Method of Holmgren.**—This consists in testing the power of a person to match various colors, conveniently used in the form of colored yarns. The set of worsteds contains three large test-skeins, namely: (1) *light pure green*, (2) *rose-purple*, (3) *red;* and 150 small skeins of the following colors: red, orange, yellow, yellow-green, pure green, blue-green, blue, violet, purple, pink, brown, and gray. In addition, there are several shades of each color, and a number of gradations of each tint, from the deepest to the lightest.

This test in use for many years is obviously unsatisfactory in several particulars and has been largely replaced by other methods.

2. **Jennings' self-recording test** is virtually the Holmgren test arranged in a different form, with small patches of colored worsteds, so placed that they are protected from dust and light, and the candidate makes his own record as the examination proceeds by pricking the record sheet with a stylus or pointed pencil. Jenning's test was the official test for color-blindness in the medical service of our army aviation corps during the war and was found to be convenient and satisfactory.

3. **Method of Thomson.**—The late Dr. William Thomson devised the following arrangement of the yarns:

> The set consists of a large green and a large rose test-skein, and 40 small skeins, each marked with a bangle having a concealed number, extending from 1 to 40, placed in a double box, so arranged as to keep the two series apart.
>
> The large green skein being placed near by, the small skeins from 1 to 20 are placed in good daylight, and the employee under examination is directed to select 10 shades of the same color as the test-skein. One with normal vision will choose promptly the 10 green with odd numbers.
>
> A color-blind person will hesitate, and his selections will contain some even numbers, and the confusion colors will be shades of brown, etc., containing some red, or shades of gray, and will indicate the color defect. These figures are to be recorded on a blank, and the 20 skeins are to be removed. The large rose skein is then used, and the examination repeated in like manner with skeins numbered from 21 to 40, and the result recorded. The confusion skeins, which have even numbers, are blue, green, and gray. From the selections made by the man found color defective by the green test we are able to decide the character of his color-blindness. Those selecting blues are red-blind, those taking greens and grays are green-blind, according to the nomenclature of Holmgren. There are 10 roses and 10 confusion colors in the second series.

4. **The Lantern Test.**—To control and also to substitute the various wool-tests, lanterns for detecting color-blindness are employed. Useful models have been designed by William Thomson, Charles H.

Williams, and Edridge-Green. Concerning lantern tests, Dr. Thomson wrote as follows: "Whilst the wool-tests have been accepted universally as requisite for the detection of color defects, the employees of railroads and their friends have always objected to their use as having no relation to their daily duties, and have demanded such colors as are employed as signals. Furthermore, for two-fifths of the time during the night of an employee's life he is expected to govern his actions by colored lights, and hence a lantern which can imitate the night signals in form, color, intensity, and size, as they appear under all obstructions caused by rain, snow, fog, and smoke, is desirable. Its power over the wools to detect the central amblyopias of tobacco, alcohol, drugs, and disease, that would not be revealed by the skeins, make it a necessity."

Dr. Thomson described his lantern as follows: "It consists of an asbestos chimney, which can be placed on the kerosene lamp in universal use on railroads, or, over an Argand or other gas light, electric lamp, or spring candle-stick. Two disks 4 inches in diameter are so placed upon the chimney as to permit of their being superimposed partly. The lower disk contains seven glasses in apertures $\frac{1}{2}$ inch in diameter, having the white, red, green, and blue colors in general use on railroads. This may be considered the 'examination in chief,' whilst the upper disk, when combined with the lower by turning one or both, furnishes the 'cross-examination.' The upper disk has two apertures, one $\frac{1}{12}$ inch, the other $\frac{1}{2}$ inch, with white glass. The other five have one white ground glass, one deep London smoke, one pink, one green, and one cobalt-blue glass.

"The combination of the white ground and the smoke glass with the reds and greens of the lower disk enables all atmospheric conditions to be imitated, and the lights to be diminished in brightness and tint. The use of the small opening enables size and distance of signals to be imitated.

"The standard for color-sense is taken as an opening of $\frac{1}{12}$ inch at 20 feet. A man failing to see the colored light at this distance may have it increased ten times $= \frac{20}{200}$ by using the large openings. Again failing, he may approach to 1 foot and reveal a color-sense equal to $\frac{1}{200}$ only. The resemblance to the tests for form by Snellen's letter is to be noted. Since the color-blind depend alone on intensity of brightness to distinguish the white, green, and red signals, the diminishing effect of the ground glass and of the London smoke often reveals the defect. The cobalt, transmitting blue and red both, is usually described by the color-blind as blue, which color they always see well, being blind for red. The cobalt, combined with the lower reds, gives a very deep red color, which, when compared with the usual red, may induce the color-blind to name one red, the other green. Combined with the lower blue it gives a deep pink, called blue by the color-blind.

"In the pink, London smoke, and light green glasses in the upper disk I have imitated the 'confusion colors.' The pink looks cherry red to the normal eye, but it transmits both red and blue by the spectroscope, hence the color-blind pronounce it blue, or, when backed by a yellow flame, white. The light green is also called white, as is also the light gray of the London smoke. Hence we have in these three glasses tints which the color-blind name white, and reveal their defect thereby.

"The upper disk has its seven openings marked by the letters of the alphabet, and the lower by the numerals from one to seven. The examination should be made in a darkened room, and the results reported on a blank, the details being used when requisite. The man examined is expected to call or name the colors and to recognize them at 20 feet."

5. **The Pseudo-isochromatic Plates of Stilling.**—These consist of a series of plates (10 in number), each plate containing 4 squares

filled by small, irregular, colored spots, among which other spots in a confusion color, made to conform to an Arabic figure, are placed. The test-plate is held in a good light, and the examiner requires the subject to distinguish the tracings. Dr. Shinobu Ishihara of Tokyo, on the same principle as the Stilling tests, has constructed a convenient series of plates for testing color-blindness.

6. **Nagel's Card Test.**—This test consists of a set of cards on each one of which a series of colored disks arranged in a ring is placed. On some of the cards all of the disks are of one color; on others the disks are of two or three different colors, that is, confusion colors. The cards being displayed in a good light the candidate is required to state which rings are monochromatic and is next required to select in the dichromatic or trichomatic rings all of the disks which are of one special color. The results demonstrate whether the candidate is color-blind and also to which class of the color-blind he belongs.

With no single test for color-blindness is infallible, good results can with care be obtained. Parsons believes that "a properly constructed lantern will eliminate all bad cases." Duane recommends Nagel's or Stilling's test, supplemented by the lantern test, as the most satisfactory means of detecting color-blindness. In this country in practical work a combination of the wool and lantern tests is commonly employed; certainly the wools should not be used to the exclusion of other tests, especially those which include a suitable lantern.

7. **Special Tests.**—These include the use of the *spectroscope* and various forms of *chromatometers*, for example, the chromatophotometer of Chibret. For an accurate determination *Nagel's anomaloscope* may be employed. The instrument consists essentially of a mounted telescope tube into which the observer looks and sees two semicircles, one illuminated with sodium yellow and the other either with lithium red, or thallium green, or a mixture of red and green. The color intensities can be varied by a measured amount, and those used to make a match between the two semicircles indicate the character of the particular color-sense.

Direct vision for colors may be studied by placing the patient at a given distance from a chart or disk of graduated colors, and noting the amount of surface exposure which is required for the color to be properly recognized. In the scale of De Wecker and Masselon the colored surface, 2 cm. square, should be recognized at 5 meters; that is, the chromatic vision or V.C. or C. = 1; if a colored test must be four times this size in order to be recognized, C. = $\frac{1}{4}$, etc. Snydacker affixes to the ordinary type card squares of red (2.5 and 5 mm. in size) and green (3 to 6 mm. in size), the test being made at 6 meters. Failure to differentiate the larger test objects suggests the need of perimetric examination; also central scotomas for red and green may be thus rapidly detected (see page 91).

Accommodation has been defined to be those changes in the optical adjustment of the eye effected by the ciliary muscle, and in practice is measured by finding the nearest point at which fine print can

be clearly deciphered, or by Duane's test (see page 39). The type usually adopted is that known as Snellen's 0.5 or Jäger's 1 (see also page 36).

In order to study the phenomena of accommodation the student should record: (1) The nearest point of perfectly distinct vision attainable with the smallest readable type, or the *punctum proximum* (abbreviated *p. p.*, or simply *p.*). (2) The farthest point of distinct vision, or *punctum remotum* (abbreviated *p. r.*, or simply *r.*). (3) The *amplitude* of accommodation, or the expression of the amount of accommodative effort of which the eye is capable. This is expressed in the number of that convex lens, placed close to the cornea, whose focal length equals the distance from the near point to the cornea, and which gives rays a direction as if they had come from the far point; thus, if the near point be 7 cm., the lens which expresses the amplitude of accommodation is $+ 14\text{D} \frac{100}{7} = 14$. (4) *Relative accommodation*, or that independent portion of this function which can be exercised without alteration in a given amount of convergence, and which is divided into a *negative* portion, or that portion which is already in use, and a *positive* portion, or that portion which is not in use (see also pages 35–41 and 47).

Mobility of the Eyes.—This is tested by causing the patient to follow with his eyes, the head remaining stationary, the movements of the uplifted finger, which is directed to the right, to the left, upward and downward; or, better, a small electric light, the reflections of which from the cornea can be noted. Both eyes must be observed, and note made of any lagging in their movements, or of the failure of either eye readily to turn into the nasal or temporal canthus. At the same time the relation of the movements of the upper lid to those of the eyeball is recorded. The attention of the patient must be centered upon the moving test, and allowance should be made for the imperfect mobility of highly myopic eyes. Any asymmetry of the skull or difference in the level of the two orbital margins may be observed, because such conditions are not infrequently associated with ametropic eyes, especially where the two eyes possess great inequality in refractive conditions. (For accurate determination of ocular rotations see page 603 and Appendix, page 801.)

Balance of the Exterior Eye Muscles.—Under normal conditions perfect equilibrium of the exterior eye muscles is present, and there is no interference with binocular fixation and binocular single vision (see page 602). Under abnormal conditions the movements of the eyes may be deranged so that one eye deviates, or tends to deviate, from the point of fixation—that is, from the object which it is regarding. These deviations may be classified thus:

1. *Manifest deviation*—that is, a deviation of an eye which the patient cannot overcome. This is known as *strabismus, squint*, or *heterotropia*, and is fully considered on pages 604 and 605.

2. *Latent deviation*—that is, a tendency of the visual line to deviate from the point of fixation. This tendency, however, is overcome by a

muscular effort, owing to the stimulus which the eyes always have to maintain binocular single vision. It is generally described by the term *latent squint, suppressed squint,* or *heterophoria.* It is frequently designated *insufficiency of the ocular muscles,* and was called by von Graefe *dynamic strabismus* (see page 638).

According to the late G. T. Stevens, the various conditions of equilibrium or variation from it may be arranged in four classes:

1. *Orthophoria,* a tending of the visual lines in parallelism.
2. *Heterophoria,* a tending of the visual lines in some other direction, but with ability to adjust them habitually for single vision.
3. *Heterotropia,* a deviation of the visual lines from parallelism in such manner that they cannot habitually be united at the same point of fixation.
4. *Anotropia, katotropia;* or *anophoria, katophoria*—variations from equilibrium which may or may not be consistent with parallelism of the visual lines, but in which, with the least innervation of the eye muscles, the visual lines of both eyes would fall below (katotropia) or rise above (anotropia) the most favorable plane for the minimum effort. Thus with ano- or katotropia there may be associated heterophoria or heterotropia.

Heterophoria may be divided into the following specific conditions:

1. *Esophoria,* a tending of the visual lines inward.
2. *Exophoria,* a tending of the visual lines outward.
3. *Hyperphoria* (right or left), a tending of the visual line of one eye (right or left) in a direction above its fellow, constituting, as the case may be, right or left hyperphoria.

The term does not imply that the line to which it is referred is too high, but that it tends higher than the other, without indicating which may be at fault.

The compound tendencies are:

1. *Hyperesophoria* (right or left), a tendency of one visual line to rise above the other, with a tendency also of the lines inward.
2. *Hyperexophoria* (right or left), a tendency of one visual line to rise above the other, with a tendency also outward.

Heterotropia may be divided into two subclasses:

(*a*) Deviations consistent with a physiologic state of the muscles and nerves, as in the ordinary concomitant squint.

(*b*) Deviations resulting from pathologic conditions—as, for example, deviations from paralysis or from mechanical causes.

The specific divisions of the subclass (*a*) are:

1. *Esotropia,* a deviation of the visual lines inward.
2. *Exotropia,* a deviation of the visual lines outward.
3. *Hypertropia* (right or left), a deviation of one visual line above the other.
4. *Hyperesotropia* and *hyperexotropia* are the compound deviations.

Cyclophoria is a term introduced by Savage to describe want of equilibrium of the oblique muscles.

In order to ascertain the condition of the ocular muscles the following tests are employed:

1. **The Screen (Cover) and Parallax Tests.**—Require the patient to regard a small point of light upon a black background 5 or 6 meters distant, or a round black spot 1 inch in diameter in the center of a white cardboard at a similar distance. Cover the left eye with a screen, making sure that the patient is fixating the test-object with his right eye. Pass the screen rapidly from the left to the right, and observe the movements of the eye which take place behind the cover. Outward deviation indicates exophoria; inward deviation, esophoria; vertical

deviation, hyperphoria. The prism, placed base inward, which neutralizes the outward deviation is a measure of the exophoria; the prism, placed base outward, which neutralizes the inward deviation is a measure of the esophoria; the prism, placed base up or base down, which neutralizes the vertical deviation is a measure of the hyperphoria.

While the screen is being moved rapidly from one eye to the other request the patient to describe the apparent movement of the test-object. If this moves in the same direction in which the cover is moved, exophoria is indicated; if in the opposite direction, esophoria; if upward or downward, hyperphoria. These apparent movements may be neutralized as before with appropriately placed prisms.

Require the patient to fix upon a fine object, as a pencil-point, held below the horizontal, 20 or 25 cm. from the eye, and, in order to remove the control of binocular vision, cover one eye with a card or the hand, and observe whether the eye under cover deviates inward or outward, upward or downward, and returns to fixation when the cover is removed, and neutralize the movement with appropriately placed prisms.

2. **Prism Tests.**—A small flame is placed against a dark background at 5 or 6 meters from the patient, and on a level with his eyes. In an accurately adjusted trial-frame a prism of 7° is inserted, base down, before one eye—for example, the right. Vertical diplopia is induced, and the upper image belongs to the right eye. If the flames stand one directly over the other, there is no inclination to divergence or convergence. If the upper image stands to the left, there is exophoria; if to the right, esophoria. That prism placed with its base in or out before the left eye, according to circumstances, which brings the two images into a vertical line, measures the degree of the deviation.

In order to test the functions of the vertical muscles at a distance of 6 meters the patient is seated as before, and a prism of sufficient strength to induce homonymous diplopia is placed before one eye—for example, the right—*i. e.*, with its base toward the nose. If the images are on the same level, no deviating tendency is present. If, for example, the right image rises higher than the other, the visual line of the right eye tends to be lower than that of its fellow, and there is hyperphoria. That prism, placed with its base down before the left eye, which restores the images to the horizontal level, measures the degree of deviation.

3. **Equilibrium Test.**—In order to test the functions of the lateral muscles at the ordinary working distance, or 30 cm., it is customary to employ the equilibrium test of von Graefe, in which a card, having upon it a large dot, through which a fine line is drawn, is held 25 or 30 cm. from the eyes, diplopia being induced by means of a prism of 10°, base up or down, before one eye. A more accurate test-object is a small dot and fine line, or a singe word printed in fine type, requiring accurate fixation and a sustained effort of accommodation. If, the prism being placed base down before the right eye, the images stand exactly one above the other, equilibrium is evident; if the upper image (image of the right eye) stands to the left of the lower image, there is *crossed*

lateral deviation; and that prism, placed before the left eye with its base toward the nose, which restores the images to a vertical line, measures the tendency to divergence or exophoria. If the upper image stands to the right of the lower, there is *homonymous lateral deviation;* and that prism, placed before the left eye with its base toward the temple, which restores the images to a vertical line, measures the tendency to convergence or esophoria. The vertical muscles should also be tested at the ordinary working distance.

Edward Jackson's test for muscle balance at the working distance is useful. It consists of a small white square on a black ground, which is held at a distance of 33 cm. from the eyes, and which is regarded through a strong convex cylinder (10 or 12 D) placed before one eye. The white spot appears as a gray streak, which seems to pass through the spot if there is orthophoria, but to one or other side of it, or above or below it, if there is heterophoria. The axis of the cylinder must be vertical to test the vertical balance, and horizontal to test the lateral balance. The Maddox rod test (see page 81) has also been adapted to the working distance, the test-object being a small illuminated square on a black ground, which is viewed at the near point through a Maddox rod. Schild, B. F. Baer, Jr., and James Thorington have designed useful instruments. The Maddox *wing test* is most satisfactory. The instrument is used for testing all varieties of heterophoria at close (33 cm.) ranges. A descriptive pamphlet accompanies the instrument.



Fig. 32.—Risley's rotary prism.

4. In order to test the **convergence near point,** approach a finger or a pencil to the nearest point upon which the eyes can converge. This should be situated at no greater distance than 8 cm. ($3\frac{1}{2}$ inches) from the eyes—that is, 2.5 to 4.5 cm. ($1–1\frac{3}{4}$ inches) from the nose. If, before this point is reached, outward deviation of one eye occurs, the amount of convergence is deficient (see also page 602).

5. In order to ascertain the power of **adduction** (properly *prism-convergence*), **abduction** (properly *prism-divergence*), and **sursumduction** (*sursumvergence*), the strongest prism which the lateral and vertical muscles can overcome is found (see page 603).[1]

Beginning with *adduction* (*prism-convergence*), find the strongest prism placed before one eye, with its base toward the temple, through which the flame still remains single. The test should begin with a weak prism, the strength of which is gradually increased until the limit is as-

[1] According to Duane, the term "adduction" is properly applied only to the amount (40°–50°) by which each eye can turn inward when moving parallel with its fellow (*associated adduction*, or adduction proper); "abduction," to the absolute degree of rotation of each eye outward in performing associated parallel movements, which is 40°–50°; "sursumduction," to the absolute degree of movement of either eye upward—a movement of some 40° in extent.

certained. This varies from 30° to 60°, the higher degrees, however, in most instances being attained only after a reasonable degree of practice.

In like manner *abduction* (*prism-divergence*) is tested, the prism now being turned with its base toward the nose; 6° to 8° of prism should be overcome. The ratio between adduction and abduction should be 6 to 1 (Stevens)—*i. e.*, if adduction is 48°, abduction should be 8°; but, according to Risley, in carefully corrected or emmetropic eyes, the ratio is 3 to 1. Banister found the primary adduction for 6 meters to be only 14°. As Hansell and Reber properly observe, no arbitrary standard of the ratio between prism-convergence and prism-divergence can be given, although the latter is fairly constant under normal conditions.

Sursumduction (*sursumvergence*), or the power of uniting the image of the candle-flame, seen through a prism placed with its base downward before one eye, with the image of the same object as seen by the other eye, is ascertained by beginning the trial with a weak prism, $\frac{1}{2}°$ or 1°, and gradually increasing its strength. The limit is usually 2°,

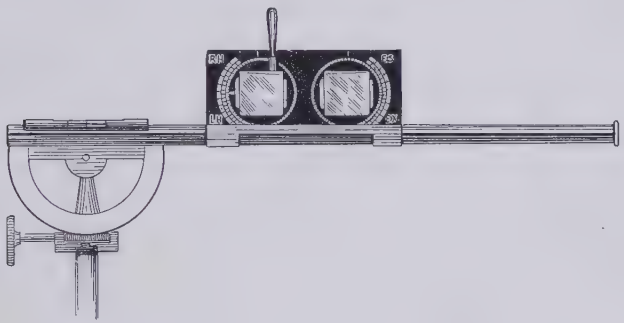


Fig. 33.—Stevens' phorometer.

but may be as high as 6° or 8°. *Right sursumvergence* is equivalent to the degree of that prism placed base down before the right eye (or base up before the left), and *left sursumvergence* to the degree of that prism placed base down before the left eye (base up before the right) through which the test-object still remains single. Right and left sursumvergence are normally equal.

If the eyes of the patient under examination are ametropic, the proper correcting lenses should be placed before them, and the examination for the various forms of heterophoria made through this glass, which should be accurately centered.

Practically all the examinations for muscular errors can be made with a series of prisms and a trial-frame, but they are facilitated by the use of certain instruments of precision, especially some form of Herschel or *revolving prism*, the one devised by Risley being one of the best. The latter consists of two prisms, superimposed with their bases in opposite directions, constituting a total value of 45°. They are mounted in a cell which has a delicately milled edge, and fits in the or-

dinary trial-frame. The milled edge permits convenient turning in the frame, so that the base or apex of the prisms can be readily placed in any desired direction. The prisms are caused to rotate in opposite directions by means of a milled screw-head, projecting from the front of the cell. With this rotary prism the strength of the abducting, adducting, and supra- and infraducting muscles can be measured. If the rotary prism is placed before the left eye with the zero mark vertical and the screw turned to the right or left, it will cause the base of the resulting prisms to be either inward or outward—that is, toward the nose or temple, as may be desired; or it may be placed with the zero mark horizontal, and the base turned upward or downward. All examinations for muscular defects may be made with a *phorometer*. A number of excellent models are available.

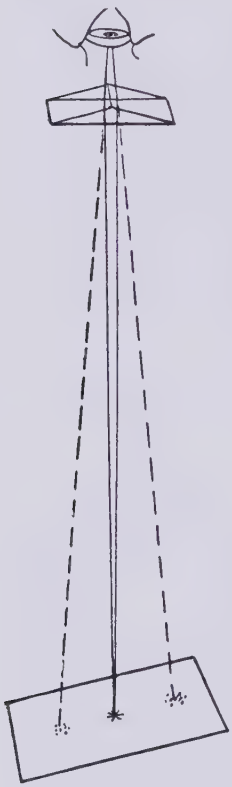


Fig. 34.—Position of the images as seen through the obtuse-angled prism of Maddox.

6. **Obtuse-angled Prism Test.**—One of the simplest tests of the ocular muscles is the *obtuse-angled prism* of Maddox. This is composed of

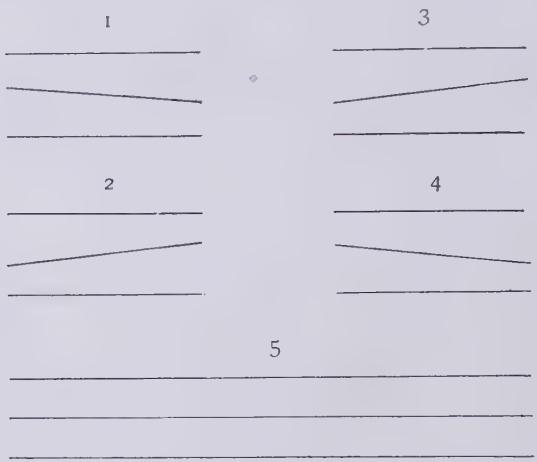


Fig. 35.—Tests for insufficiency of oblique muscles: 1, Insufficiency of left superior oblique; 2, insufficiency of left inferior oblique; 3, insufficiency of right superior oblique; 4, insufficiency of right inferior oblique; 5, equilibrium of oblique muscles (Savage).

"two weak prisms of 3° united by their bases. On looking through the line thus formed at a distant flame, two false images of it are seen, one higher and one lower than the real image seen by the other eye, the position of which, to the right or the left of the line between the false images, indicates the equilibrium of the eye. A faint band of light, of the same breadth as the two false images, is seen extended between them" (Fig. 34). The answers of the patient may be materially assisted by placing a red glass before one eye, and thus tinting the real image.

If this stands directly in the center between the two false images, all forms of latent deviation are eliminated; if it stands to the right or to the left, there is exophoria or esophoria; if it stands above or below the center, or is fused with either the upper or the lower image, there is hyperphoria.

7. **Cyclophoria** (*insufficiency of the oblique muscles*), according to Savage, may be detected "by placing a Maddox prism, with its axis vertical, before one eye, the other being covered, which regards a horizontal line on a card 18 inches distant. This line appears to be two, each parallel with the other. The other eye is now uncovered, and a third line is seen between the other two, with which it should be parallel. Want of harmony in the oblique muscles is shown by want of parallelism of the middle with the other two lines, the right end of the middle line pointing toward the bottom and the left end toward the top line, or *vice versa*, depending upon the nature of the case" (Fig. 35). The Maddox wing-test provides a method for determining the presence of cyclophoria which is very satisfactory (see also page 78).

8. **Cobalt Test.**—A trial-frame armed on one side—for example, the right—with a piece of *cobalt glass* is placed in position and the patient required to regard the test-light. The right image will be smaller than the left, and have a blue center and a red border if the patient is hyperopic or emmetropic, and a red center with a blue border if the patient is myopic. Suitably placed prisms, which unite the images, are the measures of the deviation.

9. **The Rod Test.**—This test was designed by Maddox, and depends upon the property of transparent cylinders to cause apparent elongation of an object viewed through them, so that a point of light becomes a line of light so dissimilar from the test-light that the images are not united. It may be suitably employed by having mounted in a cell, which will fit in the trial-frame, a transparent glass rod colored red, $\frac{3}{4}$ inch long, and about the thickness of the ordinary stirring rod used by chemists, or a series of glass rods placed one above the other (Fig. 36).

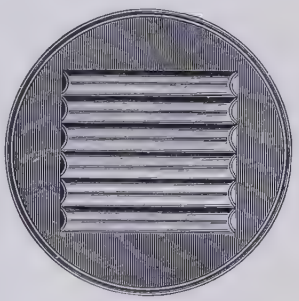


Fig. 36.—Maddox multiple rod.

The examination for *horizontal deviation* is thus described: "Seat the patient at 6 meters from a circle of light 5 mm. in diameter, and place the rod horizontally before one eye. If the line passes through the circle of light, there is orthophoria (equipoise), as far as the horizontal movements of the eyes are concerned. Should the line lie to either side of the circle of light, as in most people it will, there is either latent convergence or latent divergence; the former, if the line is on the same side as the rod (homonymous diplopia); the latter, if on the other side (crossed diplopia)."

In order to test the *vertical deviation* the rod is placed vertically

before the eye; a horizontal line of light appears, and the patient is asked if the line passes directly through the flame or if it appears above or below it. The following rule, quoted from Maddox, will suffice to indicate the "hyperphoric" eye: "If the circle of light is lowest, there is a tendency to upward deviation of the naked eye; if the line is lowest, of the eye before which the rod is placed."

The measurement of the extent of the deviation may be made in the ordinary way by finding that prism, placed before the naked eye

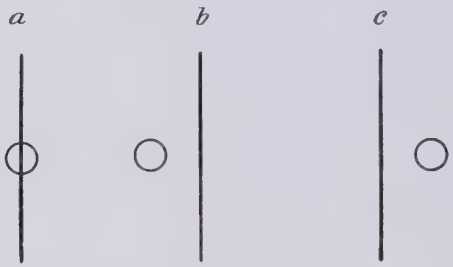


FIG. 37.—Maddox's rod test for horizontal deviation. The rod is before the right eye. *a*, The line passes through the circle of light—orthophoria. *b*, The line passes to the right of the light—latent convergence, or esophoria. *c*, The line passes to the left of the light—latent divergence, or exophoria.

(preferably with the rotary prism of Risley), which brings the line and flame together.

Of the various tests described, the Maddox rod is simple, and for all practical purposes accurate, especially when it is employed to estimate vertical deviations. According to Duane, it is apt to indicate an excess of deviation, particularly in esophoria. To obviate this fault P. Dolman suggests a combination of the screen and Maddox rod test

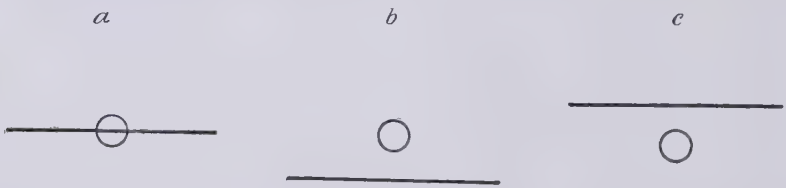


FIG. 38.—Maddox's rod test for vertical deviation. The rod is before the right eye. *a*, The line passes through the circle of light—orthophoria. *b*, The line passes below the light. The upper image belongs to the left eye—right hyperphoria. *c*, The line passes above the light. The upper image belongs to the right eye—left hyperphoria.

(*Maddox rod-screen test*). Hansell and Reber doubt if the prism-test reveals the true state of the muscle balance. They have found distinct contradictions between its results and those of the Maddox rod and other tests, and this is a matter of common experience. The screen and parallax tests, if carefully and repeatedly performed, give, as Duane has demonstrated, most trustworthy information.

Amplitude Convergence.—In order to determine the maximum of convergence, an instrument known as an *ophthalmodynamometer* may be employed. The one devised by Landolt,[1] consists of a metallic cylinder, blackened on the outside, placed over a candle-flame. The cylinder contains a vertical slit, 0.3 mm. wide, covered by ground glass. The luminous vertical line thus produced is the object of fixation. Beneath the cylinder is attached a tape-measure graduated on one side in centimeters, and on the other in the corresponding number of meter angles. The fixation object is gradually approached in the median line toward the patient, until that point where double vision occurs is reached, or the nearest point (*punctum proximum*) of convergence, and the distance in centimeters read from one side of the tape, and the corresponding maximum of convergence in meter angles on the other.

The minimum of convergence may also be ascertained with the instrument, but when this is *negative* it is determined by finding the strongest abducting prism—that is, base in before one eye—which will not cause diplopia while the patient is fixing a candle-flame at 6 meters. If the number of the prism is divided by 7, the quotient will approximately give in meter angles the amount of deviation of each eye when the prism is placed before one. The amplitude of convergence is equivalent to the difference between the maximum and minimum of convergence—that is, $a = p - r$. Thus, if the normal average of maximum convergence is 9.5 meter angles and the average minimum of convergence is — 1 meter angle, the amplitude of convergence would be $a = 9.5 - (-1) = 10.5$ meter angles. (See Meter Angles, page 45.)

The Field of Vision.—When the visual axis of one eye is directed to a stationary point, not only is the object thus "fixed" or "fixated" visible, but also all other objects contained within a given space, which is large or small, in proportion to the distance of the fixation point from the eye. This space is the *field of vision*, and the objects within it imprint their images upon the peripheral portions of the retina, or those which are independent of the macula lutea. In contradistinction to visual acuteness and refraction, which pertain to the macula in the act of *direct vision*, the function of sight capable of being performed by the rest of the retina is called *indirect vision*.

The limits of the visual field may be roughly ascertained in the following manner: Place the patient with his back to the source of light, and have him fixate the eye under examination, the other being covered, upon the center of the face of the observer or upon the eye of the observer which is directly opposite his own, at a distance of 2 feet. Then let the surgeon move his hand in various directions midway between himself and the patient, on a plane with his own face, until the limits of indirect vision are determined, controlling at the same time the extent and direction of the movements by his own field of vision.

In place of the hand other test objects may be employed. The author is accustomed to use ivory balls, 10 mm. in diameter, white and colored; mounted on the ends of black rods 45 cm. in length. The

[1] Landolt Refraction and Accommodation of the Eye, p. 283.

technic of examination is precisely the same as that of the "hand-test." Duane recommends as a suitable test object for this purpose a white card with a round black spot 1 to 3 mm. in diameter on each side of it, and for color tests a small colored square on each side of a gray card, the gray having the same light value as the color which it bears. These methods, *confrontation methods*, to use Duane's descriptive term, furnish trustworthy results. Naturally in the event of the discovery of any defect in the visual fields they must be measured by the more exact procedures of perimetry.

If it is desired to have a map of the field not larger than 45° in extent, let the patient be placed 25 cm. from a blackboard, which may be conveniently ruled in squares, and fixate the eye under observation upon a small white mark. The observer then moves the test-object—a piece of white or colored paper 1 cm. square, affixed to a black handle

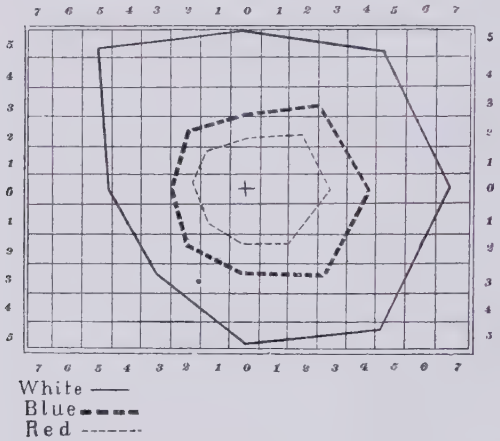


FIG. 39.—Limits of the normal field for white, blue, and red, transcribed upon a blackboard.

—from the periphery toward fixation, until the object is seen. If eight peripheral points are marked and afterward joined by a line, a fair map of the field of vision will be obtained,[1] which may be tran-

[1] The value in degrees of the squares on the blackboard may be casertained by the following table, provided the eye is placed exactly at 25 cm. from the fixation point:

2.2 cm.	=	5°	in the perimeter semicircle.	
4.4 cm.	=	10°	"	"
6.7 cm.	=	15°	"	"
9.1 cm.	=	20°	"	"
11.7 cm.	=	25°	"	"
14.4 cm.	=	30°	"	"
17.5 cm.	=	35°	"	"
21 cm.	=	40°	"	"
25 cm.	=	45°	"	"
30 cm.	=	50°	"	"
36.7 cm.	=	55°	"	"
43.3 cm.	=	60°	"	"

scribed upon a chart, like the one originally suggested by Joy Jeffries (Fig. 39).

Bjerrum's Method.—This method, proposed by Bjerrum in 1899, consists essentially in the use of white test-objects which subtend a very small visual angle, the examination to be made at a distance of 2 meters, while the eye fixates the marked center of a black screen 2 meters in breadth, which can be let down from the ceiling to the floor. In place of Bjerrum's curtain, Duane's tangent plane serves an admirable purpose. A. H. H. Sinclair uses a screen covered by heavy black velvet. Peter has designed a 1-meter screen equipped with "day-lite" filter illumination.

Bjerrum's test-objects are small, cricular disks of ivory, fixed on the ends of long, dull, black rods. They vary from 1 to 10 mm. in diameter. The examination may begin in the ordinary manner (at 30 cm.) with the 10 mm. disk and continue at 2 meters' distance with a 3-mm. disk. In the first instance the visual angle approximately is 2°, the normal boundaries of the field have been recorded. In the second instance the field boundaries are 35° outward, 30° inward, 28° downward, and 25° upward. At a distance of 2 meters, the blind spot, for example (see page 91), instead of measuring 2.5 cm., as on an ordinary perimeter, measures 20 cm. in diameter.

As ordinarily employed, the Bjerrum curtain, or its equivalent, is placed at 0.75 meter (30 inches) from the eye, if it is desired to ascertain the limits of the visual field within 50° from the center, and at 1.5 meters (60 inches) if it is desired to search for and investigate central and paracentral scotomas and enlargements of the blind spot.[1]

Among the various *campimeters* which have been designed, the *hand campimeter* of L. C. Peter[2] is a noteworthy and exceedingly satisfactory instrument. It, as Peter properly maintains, is adapted to studies within 30° of the fixation point, and a reasonable degree of accuracy can be obtained in peripheral deliminations of the field up to 40°.

From the preceding descriptions it is evident that beyond 45° the campimeter method ceases to be accurate, because on a flat surface the object is too far away from the eye; rays perpendicular to the visual line coming from a peripheral object would be parallel to the blackboard, and could not arise from it, or any object passed across its surface.

Hence, the investigation of the periphery of the retina requires the use of an instrument known as a *perimeter*. This consists essentially of an arc marked in degrees, which rotates around a central pivot, that at the same time may be the fixing-point of the patient's eye, which is placed 33 cm. distant at the center of curvature of the per-

[1] W. G. Sym and A. H. H. Sinclair have described the necessary apparatus for Bjerrum's test for scotomas in the field of vision (Ophthalmic Review, 1906, vol. xxv, p. 141); and T. B. Holloway has designed a conveniently mounted set of Bjerrum's tests, white and colored. (Transactions of the American Ophthalmological Society, 1911, vol. xii, p. 966.)

[2] Principles and Practice of Perimetry, second edition, 1923, p. 80.

imeter arc. The test-object, 5 to 10 mm. in diameter, affixed upon a carrier, is moved from without inward, and the point noted on each meridian where it is recognized. The result is transcribed upon a chart, prepared by having ruled upon it radial lines to correspond to the various positions of the arc, and concentric circles to note the degrees.

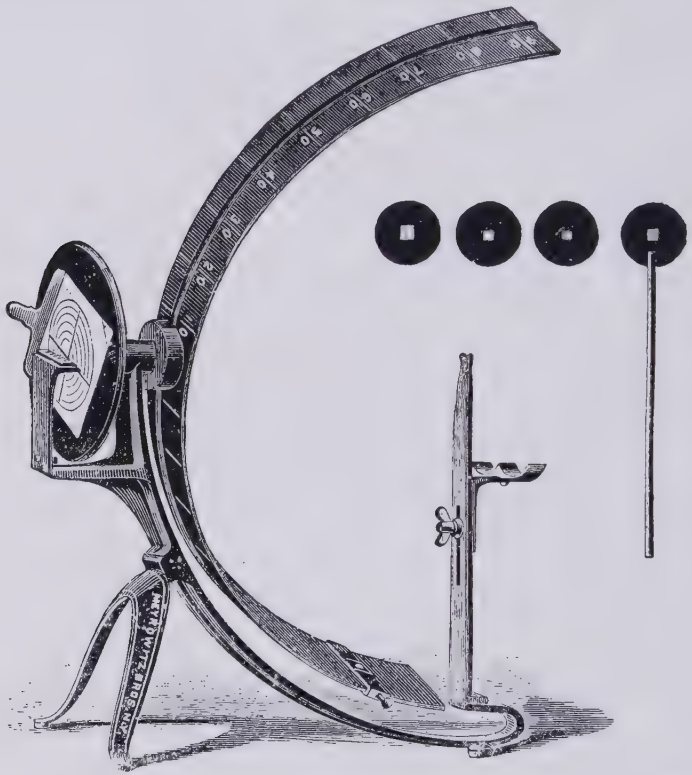


Fig. 40.—Perimeter. The examination may be made with the carrier which moves along the semicircle, or the test-objects may be carried along this by means of dark disks attached to a long handle, each disk containing in its center the test-object. The patient's chin is placed in the curved chin-rest; the notched end of the upright bar is brought in contact with the face, directly beneath the eye to be examined, which attentively fixes the center of the semicircle. The other eye should be covered, preferably with a neatly adjusted bandage. The record chart is inserted at the back of the instrument, and, by means of an ivory vernier, the examiner is enabled to mark exactly with a pencil the point on the chart corresponding to the position on the semicircle, at which the patient sees the test-object. The various marks are then joined by a continuous line, and a map of the field is obtained (see Fig. 41).

Many ingenious instruments have been devised, especially such as are self-registering, among which may be mentioned those of McHardy, Skeel, Priestley Smith, and the one recently designed by Sir William Lister. "Electric light perimeters" or "self-lit" perimeters have been designed, but they are not satisfactory. In the Ferree-Rand perimeter the electric illumination is so arranged that it remains evenly constant no matter in what position the arc is rotated around its central pivot.

This model contains a number of new features which render the instrument unusually valuable. Elliot, "dissatisfied with the limitations imposed by the flat screen, employs a perimeter of 1 meter radius, which is adapted to work either in the dark or by reflected light."

Self-registering perimeters and a sliding object holder do not achieve results which are as satisfactory as those obtained with test-objects attached to a slender black rod or wire which is moved by hand along the arc of the perimeter in the same manner as they are over the surface of a screen or blackboard; also it is important to carry the test-object not only from without inward, but, as Elliot insists, in a series of concentric circles around the fixation point, in order to detect defects in such circle. It would be an advantage, as Basil Lang maintains, if a standard rate of movement of the test-object could be established. It is necessary that the patient shall maintain a steady fixation. The hand perimeter of Schweigger for bedside examinations, and the more elaborate one of Wildbrand, which can be adjusted while the patient lies upon his back, are useful instruments.

The physiologic limits of the field for a white test-object 10 mm. in width, the eye being 33 cm. from the fixation point, are: outward, 90°; outward and upward, 70°; upward, 50°; upward and inward, 55°; inward, 60°; inward and downward, 55°; downward, 72°; downward and outward, 85°.[1]

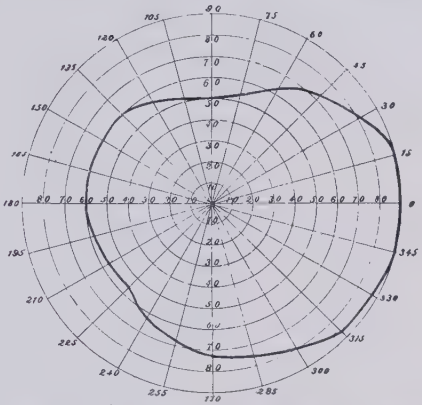


FIG. 41.—Diagram of the field of vision for white (10 mm. square test-object), transcribed upon a perimeter chart.

These measurements, which represent the *relative visual field*, vary within normal limits, and, transcribed upon a chart, produce the following figure (Fig. 41).

From this it is evident that the field of vision is not circular, being greatest in extent outward and below, and most restricted inward and above. This restriction is partly due to the presence of the edge of the orbit and the nose, and partly, as Landolt has pointed out, because the outer part of the retina is less used than the inner, and its functions, therefore, are less developed. Hence, as each portion of the field corresponds to the opposite portion of the retina, the inner part is smaller than the outer.

Quantitative Perimetry.—It must be remembered, however, that the size of the field of vision varies with the size of the visual angle subtended by the test-object with which the examination is conducted, and for the sake of accuracy the size of the object should be stated as well as

[1] Baas finds the average result of ten observers as follows: Outward, 99°; upward, 65°; inward, 63°; downward, 76°.

its distance from the patient's eye. Moreover, the visual field should not be charted with test-objects of one size only, but with a series of objects of graduated sizes. The author uses with satisfaction a series of disks mounted on long black rods, the diameters of the disks varying from 1 mm. to 4 cm. As Rönne, quoted by Traquair, says: "An examination of the field of vision in which only one object is used is in itself just as inadequate as an examination of visual acuteness with a test-card which has letters of only one size."[1]

Binocular Field of Vision.—The field of vision for each eye having been defined, it remains to point out that the field of vision which pertains to the two eyes, or that portion in which binocular vision is possible, constitutes only the area where the central and inner parts overlap. This is evident from the diagram. The continuous line *L* bounds the field of vision of the left eye, and the dotted line *R* the visual field of the right eye. The central white area corresponds to the portion common to both eyes, or to that area in which all objects are seen at the same time with both eyes; the shaded areas correspond to the portions in which binocular vision is wanting. In the middle of the

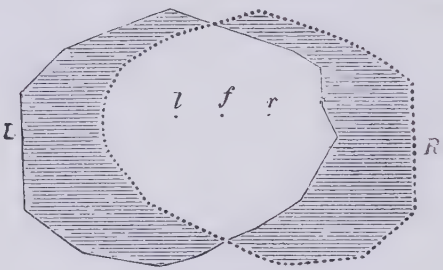


FIG. 42.—Binocular field of vision (Moser).

white area lies the fixation point *f*, and on each side of it the blind-spots of the right and left eye, *r* and *l*.

[1] H. M. Traquair (Ophthalmic Review, 1914, vol. xxxiii, p. 65) gives an excellent account of the *quantitative method* in perimetry.

Clifford B. Walker, in his examinations of patients with intracranial lesions in Harvey Cushing's service, found it necessary to investigate the field for test-objects varying in visual angle from 1 or 2 minutes to about 8°, and has designed a series of nine circular rimless colored and white disks supported on wire handles which cover the required range, taking the "normal" or 5-mm. disk as the unit. In this series the smallest disk is 0.15 mm. and the largest 4 cm. in diameter. His special perimeter has a radius of 28.6 cm. and a large working surface 60° by 130° in extent.

Col. Elliot deprecates the use of 10 or even 5 mm. disks as being unnecessarily large. According to him, with a daylight perimeter of 330 mm. radius the test-object used need be no larger than between 2.6 and 2.8 mm. in diameter, because "a full normal field for a white object can be obtained from a healthy eye if we use that object of such a size that its diameter subtends an angle of half a degree at the nodal point." Naturally, if the dimensions of the object used are reduced, the size of the field will be decreased.

To make record of the size of the test-object and the distance of the eye from the perimeter A. H. H. Sinclair suggests that the size shall be the numerator of a fraction, the denominator of which shall be the distance, both being expressed in mm., for example: visual fields obtained by test $\frac{1}{2000}$, $\frac{6}{2000}$, $\frac{10}{300}$.

Having thus determined the *limits* and *continuity* of the visual field, the functions of the peripheral parts of the retina in regard to perception of colors, acuteness of vision, and appreciation of light should be investigated.

The *color-field* is charted in the manner described in connection with the general visual field, the squares or circles of white being replaced by similar colored objects.

The order in which the colors are recognized from without inward is: (1) Blue; (2) yellow; (3) orange; (4) red; (5) green; (6) violet. In practical work, blue, red, and green are the colors employed, red and green being the color-sense most usually affected in pathologic cases. Non-saturated colors are not correctly recognized when the test-object is first seen. Thus, yellow at first appears white; orange, yellow; red, brown; green, white, gray, or gray-blue; and violet, blue. The physio-

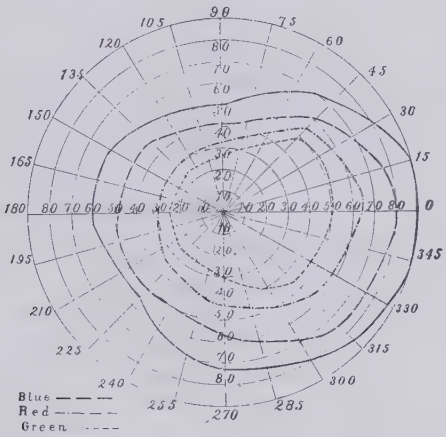


FIG. 43.—Diagram of the field of vision for blue, red, and green. The outer continuous line indicates the limit of the form-field; the broken lines, the limits of the color-fields.

logic limits of the color-fields, which, like those of the general field, are subject to variations, when estimated with a 10 mm. square test-object, the eye being 33 cm. from the fixing point, correspond closely to the following:

	Blue.	Red.	Green.
Outward	80	65	50
Outward and upward	60	45	40
Upward	40	33	27
Upward and inward	45	30	25
Inward	45	30	25
Inward and downward	50	35	27
Downward	58	45	30
Downward and outward	75	55	45

These, when transcribed upon a chart, are represented in Fig. 43.

The numbers represent the usual limits at which the color-test 10 mm. square is recognized as such. They do not indicate its greatest intensity,

which is perceived only at the fixation point. In order to avoid discrepancies, the character of the light, the nature and saturation of the color, and the size of the test-object should be stated in describing examinations. Always the visual field for colors as well as for white should be investigated with test-objects of various sizes (see also page 89).

It should be remembered that the boundaries of the color-field which have been described result from examination with test-objects not greater than 10 mm. square. With larger areas of color it will be found that the color-fields differ in extent very little from the fields for white.

The color-limits contract concentrically as the illumination is reduced, but if the photochemical and neuroptic apparatus are normal, there will be no change in the normal sequence of the color-limits. Blue should be employed in investigating defects in the photochemical apparatus, as it is the color first to disappear in reduced illumination; red suffers promptly in reduced excitability of the neuroptic apparatus.

Important as the study of the color fields is, their accurate determination is fraught with many difficulties largely owing to the failure in the standardization of color-tests. For this reason it is often the case that visual fields plotted carefully with white test-objects of varying sizes (see page 88) yield more accurate information than that derived from the color-fields (see page 89).

The *acuteness of the vision* of the *peripheral parts of the retina* may be tested with small squares of black paper (6, 5, 3, and 2 mm. black quadrants on a white ground), separated from one another by their own width, by noting the point in each meridian where they are recognized as separate objects; or with gray patches of different intensity on a white ground (Ward Holden).

The *light-sense* of the *periphery of the retina* may be tested conveniently with Ward Holden's tests. One card has a 1 mm. black point on one side and a 15 mm. quadrant of light gray, having four-fifths of the intensity of white, on the other. With a perimeter of 33 cm. radius the black point and gray patch are each seen by a normal eye outward, 45°; upward, 30°; inward, 35°; downward, 35°. The second card has a 3 mm. black point on one side and a darker gray patch, having three-fifths of the intensity of white, on the other. Each is seen on the perimeter arc outward, 70°; upward, 45°; inward, 55°; downward, 55°. Card 2 will reveal slight disturbances of light-sense near the periphery and Card 1 in the intermediate and central zones.

The *perception of light* is the most constant function of the healthy retina, and remains nearly the same throughout its surface, while the color- and form-sense rapidly lessen toward the periphery. For practical purposes, a candle-flame or small electric bulb passed along the arm of the perimeter may be used as a test-object; and, if vision is very defective, a second candle or bulb is made the point of fixation. This test does not demonstrate the light-perceiving power of the retina unless the patient is able not only to tell when it is light and when it is dark, but to indicate accurately where the light is and from what direction it is coming (see also page 461). Progressive diminution of light-sense

from center to periphery will be found if test-objects of varying luminous intensity, with the illumination of ordinary daylight, are employed.

The *adaptation of the retina* may be estimated according to Wilbrand's method of investigating the visual field in a dark room with test-object and fixation point streaked with luminous paint. The examination is made as soon as the patient enters the dark room and again in ten minutes. This interval is sufficient to enable the normal eye to adapt itself so that the extent of the visual field corresponds to that of a white object in diffuse daylight. Delayed adaptation is a phenomenon found in many pathologic conditions (see also page 560).

Abnormalities of Visual Field and Scotomas.—The most frequent departures from those limits of the visual field assumed to be normal are general or concentric contraction; contraction limited especially to one or the other side; peripheral defects in the form of re-entering angles; absence of one segment or quadrant; and absence of the entire right or left half of the field.

In addition to these defects, search should be made for dark areas within the limits of the visual field, or *scotomas*. A *positive* scotoma is one which the patient perceives as a dark spot in some portion of his visual field, especially if he regards a white surface; a *negative* scotoma, although unperceived by the patient as a dark spot in front of his eyes, is a limited area of the visual field within the confines of which an external object is not perceived and which is generally not discovered until the field is tested. Such a negative scotoma is either *absolute* or *relative*—absolute if the test-object is invisible; relative if it is simply indistinct. Thus, if a white object disappears entirely there is an *absolute scotoma for white*, but if it simply grows indistinct there is a *relative scotoma for white*. If a colored test-object (usually red, blue, or green) disappears entirely, there is *an absolute scotoma for the color used;* but if it simply grows indistinct or less vivid, there is *a relative scotoma for the color used*. Scotomas are further subdivided, according to their situation and form, into *central*, *pericentral*, *paracentral*, *ring* or *annular*, *peripapillary*, and *peripheral*.[1] A scotoma may be round or oval or of an irregular shape or assume a curved form. Important scotomas are those which are continuous with the blind-spot (see next paragraph, also page 420). A normal eye will develop a relative central scotoma in a darkened room, and if the darkness is increased the scotoma becomes absolute (Hess).

In every normal eye there is a *physiologic scotoma* corresponding to the position of the optic nerve entrance, which usually may be found 15° to the outer side of and 3° below the point of fixation; the interval, according to Landolt, being greater in hyperopic than in myopic eyes. This is known as *Mariotte's blind-spot*. It is surrounded by a narrow

[1] For a scotoma which lies between the fixation point and the blind-spot, such as occurs in toxic amblyopia (see page 564), Traquair proposes the name *centrocecal scotoma*, and for other defects of the central part of the field the terms *supracentral*, *infracentral*, *nasocentral*, *temporocentral*, and for defects adjacent to the papilla, *supracecal*, *temporocecal*, etc.

zone of relative amblyopia for white about 1° in width. According to Hansell, the average distance of the center of the blind-spot from fixation point is almost identical in emmetropia and hyperopia, but in myopia is about 5 mm. greater. Should the horizontal diameter of the blind-spot for motion exceed 6°, according to Van der Hoeve, it is larger than normal, and may indicate the presence of beginning pathologic change.

For the *delimitation of scotomas*, small test-objects, white or colored, 1–3 mm. in diameter, may be employed, which are moved in different directions from the point which the eye under observation attentively fixes, and the spot marked where the object begins to disappear or change its color. The arm of the perimeter is usually marked near the center in half-degrees for this purpose. *Scotomas* including the blind-spot, however, are best mapped out on Bjerrum's curtain or its equivalent (Duane's tangent plane) or with the aid of a campimeter, for example, Peter's, which is well adapted for the purpose. Employing the Bjerrum method, the patient should be 1.5 to 2 meters from the curtain and the test-objects 2 to 5 mm. in diameter (page 85).

Duane recommends the *complimentary color test* (*extinction test*) applied with the tangent curtain for the examination of scotomas or the blind-spots at a distance of 1.5 meters. The method is as follows: Should, for example, the left eye be under examination, it remains uncovered, while the right eye, covered with a deep amber or a ruby glass, fixates the central spot of the curtain. Next a dark blue disk on a carrier is passed in various directions along the curtain. This disk is unperceived by the right eye, because it is extinguished by the colored glass, but is visible to the left eye as a lighter blue until it enters the scotoma, when it disappears if the scotoma is absolute or becomes dim and dark.

Special instruments for detecting and measuring scotomas—*scotometers*—have been designed by Priestley Smith, P. C. Bardsley, B. T. Lang, N. Bishop Harman, and R. H. Elliot who has combined the advantages of Priestley Smith's instrument with those of Bjerrum's curtain by working at a distance of 1 meter. Haitz employs a stereoscope with diagrams which gives binocular fixation. The card before the eye to be tested is covered with small squares with which the defect is detected and mapped; each side of a square subtends an angle of 1 degree at the distance at which the card is used. *Haitz's stereoscopic charts* and the complimentary color test are especially valuable in the examination of patients whose defective central vision does not permit accurate fixation of the central spot on the curtain or on the campimeter. *Bissell's blind-spot slate*, to be used with a wide-angled stereoscope, and *Lloyd's stereocampimeter* are admirably adapted for the study of Mariotte's blind-spot and its anomalies. The stereocampimeter is most valuable in the investigation of unilateral central scotomas and defects in the central retinal area.

Visual field examinations, that is, perimetry and scotometry, constitute most important methods of functional testing in ophthalmic

work. Satisfactory results depend not so much upon the instrument and method employed, provided they are good ones, as upon the care and accuracy of the examiner, the co-operation of the patient, the character of the illumination, and the employment of suitable serial test-objects. The application of the methods of quantitative perimetry to the analysis of scotomas, as Traquair has so well shown, has greatly increased the value of this department of visual field testing.

Tension.—This term indicates the resistance of the ocular tunics, and is clinically demonstrable by palpating the globe with the finger-tips. The middle and ring-fingers are placed upon the brow of the patient, the tips of the index-fingers upon the eyeball, and gentle to-and-fro pressure made, the eyes being directed downward. This pressure must be made in such a manner as not to push the ball into the orbit; otherwise no information of its true resistance is obtained. The tension of one eye must always be compared with that of its fellow, and in any doubtful case the results may be contrasted with those obtained by examining an eye known to be normal in another patient of similar age.

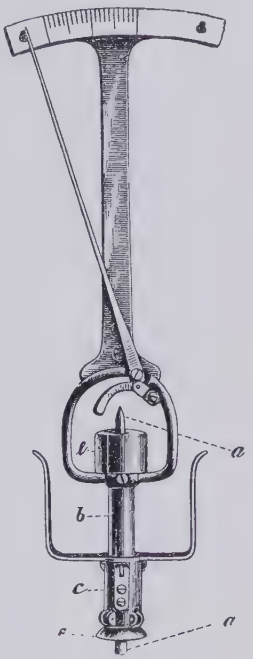


FIG. 44.—Schiötz tonometer.

Normal tension is often expressed by the sign, Tn, and the departures from it formerly were indicated by the symbols +?, +1, +2, +3, and —?, —1, —2, —3; the plus signs referred to increased, and the minus signs to decreased, resistance. In physiologic experiments various kinds of apparatus, constructed upon the principle of the manometer, are employed, and for clinical purposes instruments known as *tonometers* have been devised. The most useful of these is the one designed by Schiötz; the excursion of its pointer is indicated in millimeters (see description, page 94). A modification of this tonometer has been constructed by Gradle, and W. McLean has designed a satisfactory direct reading tonometer with constant weight factor.

The principle of the eye tonometer is thus defined by Priestley Smith: The instrument measures the impressibility of the eyeball, and from the degree of impressibility we infer the intra-ocular pressure.

The Schiötz instrument is now in common use, and its construction may be readily understood from the accompanying diagram (Fig. 44).

The patient should lie upon a table or reclining chair, with the head thrown back, and be requested to look directly upward. The eyeball is rendered insensitive with holocain (2 per cent. solution, three instillations at intervals of three minutes, or with a 2 per cent. solution of butyn), and the lids are separated without making pressure on the globe. Next, the surgeon, holding the instrument (with

the 5.5 gm. weight in place) by the two arms attached to the cuff, which slips up and down the hollow cylinder, brings its foot-piece exactly at rest on the cornea, the cuff being slid down so as to be about the middle of the cylinder. The instrument should stand freely, resting exactly on the center of the cornea. The deflection of the needle is recorded, and from a table which accompanies the instrument it may be seen with how many millimeters of mercury the deflection of the needle corresponds; thus, should the needle, with the 5.5 gm. weight in place, register 3, this corresponds to 25 mm. of mercury. Schiötz advises that three measurements shall be made and the average of the readings recorded. Some surgeons prefer a single reading taken with great care, but, as Priestley Smith insists, it is not safe to rely on a single reading; it should be confirmed by at least two additional readings. There are four weights, respectively 5.5, 7.5, 10, and 15 gm. If the needle is not deflected beyond 1, the measurement should be repeated with a greater weight. According to Schiötz, deflections between 2 and 4 mm. yield the most accurate results. The foot-piece of the instrument should be cleansed in a boric acid solution.

The normal pressure varies from 16 to 28 mm. of Hg., that is, 16 is the low and 28 the high physiologic limit, although an eye with a tension above 25 mm., 25.5 to 28, should be regarded with suspicion, should be kept under observation, and should, especially if the anterior chamber suggests shallowness, receive at night an instillation of a drop of $\frac{1}{4}$ per cent. solution of pilocarpin. According to Schiötz normal tension varies between 15.5 and 25 (22— to 40 McLean); according to Marple, between 15 and 25; according to Stock, between 12 and 26; H. Gjessing's measurements of the tension of normal eyes (2186 eyes, ages ten to eighty-seven years) yielded an average of 23.83; Criland's average (1001 examinations) was 20.06. Tensions below 13 (Schiötz) or 20 (McLean) are usually classified as subnormal. Neither the state of the refraction nor the age has an appreciable effect on the ocular tension, nor is it influenced by mydriatics, except that cocain (5 per cent.) slightly diminishes the tension of the normal eye; an effect mentioned by Heilbrun, but denied by other observers. Eserin and pilocarpin lower slightly the tension of the normal eye (Heilbrun, Gjessing, the author). Although records in the form of mm. Hg., as stated above, are usually made, as Priestley Smith points out, the reading and not the supposed equivalent in mm. Hg. should be stated (see also page 412). The readings may be recorded in the form of a fraction, for example, $\frac{5.5}{3}$. This means that with 5.5 gram weight the indicator pointed to 3 mm. on the scale. To estimate the ocular tension by means of the finger test is not a safe nor an accurate procedure, and in all circumstances where this is an important observation the tonometer should be used. Tonometric measurements are required not only if glaucoma is present or suspected, but, for example, in cases of iridocyclitis, uveitis, keratitis, especially interstitial keratitis, detachment of the retina, retinal angiosclerosis, retinal hemorrhages, and choked disk. Great care should be exercised in using the tonometer lest the foot-plate injure the cornea, especially if frequently repeated measurements are required. Abrasions of the corneal epithelium and even ulcers have been noted in such circumstances (page 314). Although holocain has no influence on the ocular tension, slit-lamp investigation

shows that it slightly affects the cornea, "polygonal dry spots" which quickly disappear, being demonstrable (Koeppe, Gjessing).

Proptosis, exophthalmos, or protrusion of the eye, may be caused by orbital diseases and tumors, sinusitis, tenotomy and paralysis of the ocular muscles, Graves' disease, and sometimes by chronic nephritis (L. Barker), and by intracranial neoplasms, especially if situated in the middle fossa of the skull; while enlargement of the ball is the result of various conditions residing within the globe—myopia, buphthalmos, intra-ocular tumor, and staphyloma. If the cause is unilateral, the resulting condition is asymmetric, and the two eyes may be compared by observing the relative positions of the apices of the corneas with each other and with the line of the brows. For measuring the degree of exophthalmos Edward Jackson has devised a simple scale or *proptometer*. A useful and accurate instrument for this purpose is the *exophthalmometer* of Hertel. A more elaborate instrument is the one designed by Lohmann. A slight protrusion of the eyeball takes place when the palpebral opening is voluntarily decidedly widened (see also page 681).

The eyeball is apparently sunken (*enophthalmos*) in some cases of ptosis, in wasting of the orbit fat, in orbital injury, and is diminished in size in high grades of hyperopia and congenital failures of development. As Nettleship pointed out, the amount of exposed sclera may help to decide the apparent protrusion or recession of the eyeball (see also page 681).

Position of the Eyes.—Instead of presenting parallel visual axes, one eye may be deviated inward, outward, downward, or upward, constituting one of the various types of strabismus (see page 604), a condition which may or may not be associated with diplopia (see page 609).

Counterfeited Blindness.—The methods for detecting malingering might be included with functional testing of the eye. They are described in Chapter XVI, p. 583, and for convenience are allowed to remain there.

CHAPTER III

REFLECTION, THE OPHTHALMOSCOPE AND ITS THEORY. OPHTHALMOSCOPY AND SKIASCOPY

Reflection.—When light falls upon a polished surface a portion of it is reflected. The angle of reflection is always equal to the angle of incidence. A polished surface, capable of reflecting light, is called a *mirror*. Mirrors are *plane*, *concave*, or *convex*.

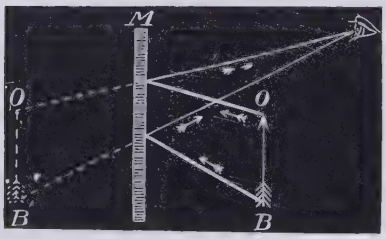


FIG. 45.—Reflection from a plane mirror.

A plane mirror reflects the rays falling upon it, so that they seem to come from a point as far back of the mirror as the object lies in front of it. It does not render the rays either convergent or divergent, nor does it lessen their convergence or divergence. Rays parallel before reflection are parallel after reflection. Rays convergent or divergent before reflection maintain the same relation after reflection. In the figure, rays from the object *O–B*, falling upon the mirror, *M*, are reflected so that they enter the observer's eye, and seem to him to come from *O′–B′*, situated as far back of the mirror as *O–B* is in front of it. The

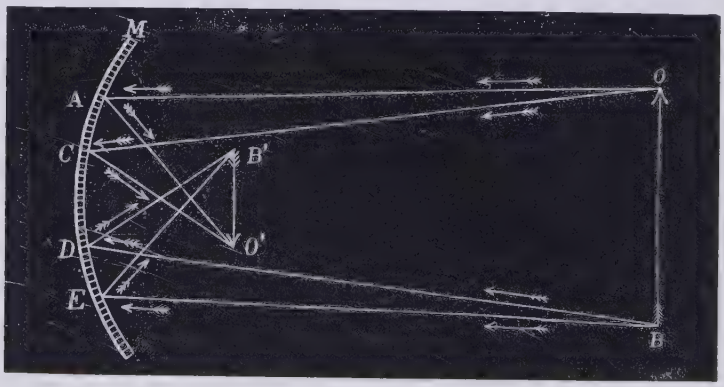


FIG. 46.—Reflection from a concave mirror.

image is not inverted. The rays have a divergence from a point whose distance is equal to the sum of the distance from the light to the mirror, and of the distance from the mirror to the eye (compare Skiascopy).

A concave mirror converges parallel rays of light to its principal focus, and forms a real, inverted image in front of the mirror.

The principal focus of a concave mirror is equal to one-half the length of its radius of curvature, $F = \frac{r}{2}$.

The conjugate focal distance for any point greater than the principal focus may be found by the following formula: f' represents the distance from which the rays diverge (the lamp or candle); f'' is the distance of the conjugate focus.

$$\frac{1}{f'} + \frac{1}{f''} = \frac{1}{F}.$$

$$\frac{1}{f''} = \frac{1}{F} - \frac{1}{f'}.$$

This may be understood by recollecting that F is the focus for parallel rays, and that the focus is the inverse of the reflective or catoptric power of the mirror. The rays which diverge from f' require $\frac{1}{f'}$ of catoptric power to render them parallel. This diminishes the catoptric power of the mirror to $\frac{1}{f''}$.

$\frac{1}{F} - \frac{1}{f'} = \frac{1}{f''}$, the focal length of f'' is the conjugate focal distance required.

Example.—The ophthalmoscopic mirror has a focus of 20 cm., its radius of curvature being 40 cm. A candle is situated at 30 cm. in front of it, and we wish to know the conjugate focal distance:

$$F = 20 \text{ cm. } f' = 30 \text{ cm. } \frac{1}{20} - \frac{1}{30} = \frac{1}{f''}, \frac{1}{f''} = \frac{1}{20} - \frac{1}{30} = \frac{1}{60}, f'' = 60 \text{ cm.}$$

The rays of the candle would be rendered convergent to a point 60 cm. in front of the mirror. The light being placed at a greater distance than the principal focus, the rays are convergent.

A convex mirror renders parallel rays divergent as if they came from its principal focus, which is *negative*, situated behind the mirror, at a distance equal to one-half the radius of curvature. The image is erect and small.

The conjugate focal distances for convex mirrors are obtained by the same formula as for concave mirrors, the sign — being prefixed to F and f''.

The cornea, by reflecting light, corresponds to a convex mirror, and in this relation is important in ophthalmometry. The principal focus of the corneal mirror is about 4 mm., the radius of curvature being 7.829 mm. The size of the image reflected from the cornea is proportional to the size of the object as the focus of the corneal mirror, 4 mm., is to the distance of the object. A candle-flame 20 mm. in diameter, situated at 100 mm., gives a corneal image whose size is found in this manner: Image : 20 :: 4 : 100. $\frac{Image}{20} = \frac{4}{100}$. Image = 0.8 mm.

If the radius of curvature is greater, the image is also greater; if the radius of curvature is smaller, the image is smaller. By this means curvature ametropia may be measured.

The size of the corneal image is so very small that it would not be feasible to attempt direct measurement of it. If two candles which are separated some distance are employed as an object, each candle represents one extremity of the object. The size of the object is, then, the distance between the two candles; the size of the image is the distance between the reflected images of the candles. Suppose this distance to be 3 mm. and by means of a double refracting prism two images of each candle are seen; if they are displaced by the prism exactly 3 mm., so that a straight line passes through all the images, two of them must overlie, as the images are 3 mm. apart. Small variations in curvature will now be manifest if the two images, which should overlie exactly, shoot past each other or fail to come together. The change of form in the crystalline lens during accommodation is proved by this experiment.

THE OPHTHALMOSCOPE

For the purpose of studying the interior of the living eye an instrument known as the *ophthalmoscope*, the invention of which, in 1851, we owe to the genius of von Helmholtz, must be employed. The original Helmholtz ophthalmoscope was composed, in general terms, of three thin glass plates, set in a suitable frame at an angle of 56 degrees to the line of sight, by means of which the light was reflected into the observed eye. With this instrument the details of the eye-ground can be studied under a weak illumination.

The modern ophthalmoscope consists essentially of a concave silvered mirror for illuminating the eye, and of lenses for measuring and modifying its refraction (*refraction ophthalmoscope*). The mirror is perforated, as originally suggested by Reute, and swings to either side, so that the obliquely incident rays may be reflected into the eye without having to tilt the entire instrument, and thus narrow the aperture and render the lenses astigmatic. The lenses are inserted in a disk, invented by Rekoss, which can be rotated in front of the sight-hole. A plane mirror, which can be substituted for the concave mirror, is a valuable addition. Many ophthalmoscopes contain two disks, which can be used either singly or in combination. This arrangement affords a series of lenses from 0.50 to 24 D concave, and from 0.50 to 23 D convex, with which the observer is enabled to view distinctly the details of the eye-ground in all forms of ametropia. A lens varying from 13 to 20 D accompanies the instrument for focal illumination of the cornea and lens, and for use in the indirect method of ophthalmoscopy. Among the many ophthalmoscopes at the student's disposal, in the author's opinion none is better than the Loring instrument (Fig. 47). A. S. Morton's ophthalmoscope is an admirable one and is much used. Excellent models have been designed by Edward Jackson, B. A. Randall, and a number of other surgeons.

Electric ophthalmoscopes, for which we are largely indebted to W. S. Dennett, are so convenient and the illumination so satisfactory both in the consulting room and at the bedside that to a certain degree they have replaced the ordinary instrument. It, however, is a mistake to use an electric ophthalmoscope to the exclusion of the other type with which the ophthalmologist and the student of ophthalmology should always be accurately familiar. The source of illumination in the electric ophthalmoscope consists of an electric bulb in the end of the handle, the light of which is condensed by a lens on a suitably tilted mirror



FIG. 47.—Loring's ophthalmoscope, with tilting mirror, complete disk of lenses from — 1 to — 8 and 0 to + 7, and supplemental quadrant containing ± 0.5 and ± 16 D. This affords 66 glasses or combinations from + 23 to — 24 D.

which reflects it into the eye. W. B. Marple's modification of Dennett's electric ophthalmoscope is, in the opinion of the author, one of the best of these instruments thus far designed (Fig. 48) and Charles H. May's ophthalmoscope is an admirable instrument. Convenient models have also been devised by S. Lewis Ziegler, George Crampton, and others.

Large demonstrating ophthalmoscopes have a distinct value in ophthalmoscopic work. The *Gullstrand ophthalmoscope* is the most perfect instrument of the type. It is free from annoying reflections and furnishes magnifications from 5 to 40 times in monocular and 20 times in binocular observations. Dr. Edward Jackson has pointed out the

advantage of *sunlight* as the source of illumination in ophthalmoscopy and also in skiascopy and transillumination. He describes the method as follows:

"Direct sunlight is the most intense light generally available for the purpose; and can be used for the examination of the eye with entire safety, by removing the heat rays, and not concentrating it too much on the tissues. In a window, through which the sun shines, a mirror is placed at such an angle as to reflect the light in the proper direction.



Fig. 48.—Marple's electric ophthalmoscope.

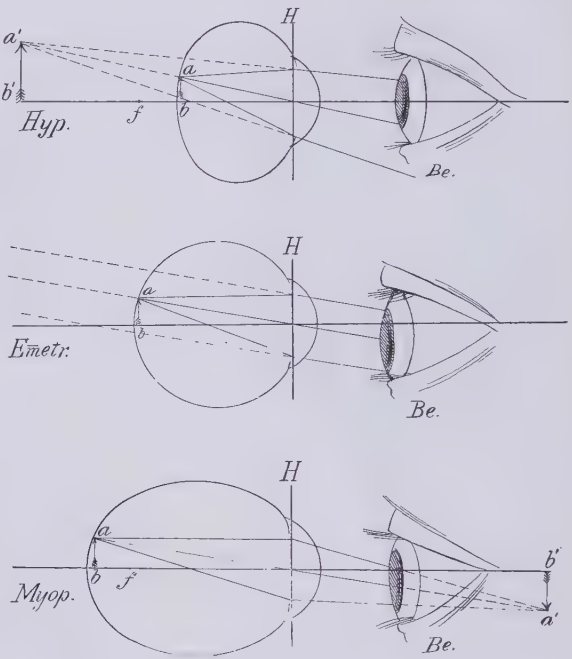


Fig. 49.—Examination of the erect image when the eye examined is hyperopic, emmetropic, or myopic. In each figure three rays are shown emanating from a luminous point on the eye-ground. In hyperopia they diverge after leaving the eye; in emmetropia they are parallel; in myopia they converge: *f*, The posterior focus; *H*, principal plane of the dioptric system of the examined eye; *Be*, observer. The ophthalmoscope is not shown (Haab).

This angle will have to be changed from time to time on account of the apparent movement of the sun in the heavens. A heliostat, in which the mirror is turned by clock work, will keep the rays properly directed as long as desired. A glass cell 20 or 25 mm. thick, containing a solution of ordinary alum, 1 to 6, is placed in the path of the light to filter out the heat rays. The light may be diffused in a diverging pencil by interposing the proper strength of lens.

"Such a diverging pencil is a convenient light for ophthalmoscopy, with the full sunlight, or rendered red-free by the necessary filters. A

diaphragm may be interposed, in which a small hole gives an excellent source of light for skiascopy, or a slit gives illumination similar to that of the slit lamp. By concentrating the light on the eye in the proper direction, it may be used for transillumination."

Direct Method.—The rays from the concave mirror, somewhat converging, enter the pupil and are brought to a focus in the vitreous humor. After reaching their focus the rays diverge again and spread out on the retina into a circle of diffusion. The portion of the retina thus illuminated sends rays back again, which pass through the dioptric media of the eye and are refracted to its far point—that is, if the eye is emmetropic, they emerge parallel and would meet at an infinite distance; if the eye is myopic, they converge to their far point in front of

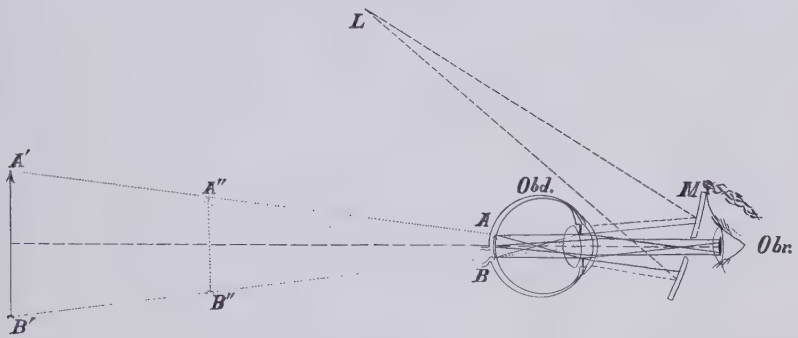


Fig. 50.—Diagram of the direct method with the formation of an upright image: Rays from the source of light, *L*, are received upon the concave mirror, *M*, and converged upon the observed eye, *Obd*, within which they cross and illuminate an area of its fundus. From an area *A–B* thus lighted rays pass out of the pupil (parallel if it be emmetropic, as here represented) through the sight-hole of the mirror, and, entering the observer's eye, *Obr*, are focused upon his retina. An image is there formed as though the object seen were at a great distance, and the perceptive centers project it into space as though the object were at some arbitrary distance (*e. g.*, 25 cm.). By the laws of magnification by a simple lens the image is embraced between the lines passing from the optical center of the magnifying lens (the refracting system of the observed eye), through the extremities of the object, and has the size *A'–B'*, *A''–B''*, etc., according to the distance of projection. In hyperopia rays from *A* and *B* would be divergent, and the observer would have to render these rays parallel by a convex glass or by using his accommodation. In myopia these rays would be convergent, and a concave glass would be required to neutralize their convergence and render them parallel. (B. A. Randall.)

the eye; if the eye is hyperopic, they diverge from their far point back of the eye (see also page 116).

An observer's eye, in order to focus these rays, must be adapted to them. If the patient is emmetropic, the observer's eye must also be rendered emmetropic. If the patient is hyperopic, the emmetropic observer must add a convex glass to his eye, or use his accommodation, in order to make the divergent rays parallel. If the patient's eye is myopic, the emmetropic observer must place a concave glass before his eye to render the convergent rays parallel. If the observer is ametropic, he must first correct his ametropia with suitable lenses (see also page 116). A hyperopic observer might see distinctly the eye of a myopic

patient, or a myopic observer might see the eye of a hyperope. In either case the hyperopia must at least be as great as the myopia.

In this method the observer sees the eye just as he would see an object through a convex glass or simple microscope. The image of the eye-ground is a virtual one—that is, it seems to be behind the eye. It is magnified and erect.

The *formation of the image* in the *direct method* may be understood by examining Fig. 50.

Divergent rays and convergent rays have been described, but always in relation to one point (see page 22). It is now necessary to consider their meaning in reference to an image.

An image is composed of a succession of points; each one of these points represents a point in the object. From the point in the object one ray passes to the optical center of the lens or lenses, and maintains the same direction after passing through it. This ray is called the *axial ray;* it passes to the corresponding point in the image. Other rays from the same point in the object diverge from the axial ray at various angles; a bundle of these rays is called a *pencil.* The size of a pencil is determined by the diameter of a lens or the aperture of the pupil. The lens gives these unequally diverging rays a direction to a common point or focus. From each point in the illuminated part of the retina a pencil of rays falls upon the crystalline lens and cornea. The size of this pencil equals the diameter of the pupil; to form an image each pencil of rays must be concentrated into one point. By diverging and converging rays is meant the relation the rays from each point bear to each other, not the relation of rays from different points.

Size of the Image.—The details of the eye-ground are considerably magnified in the direct method of examination. In the emmetropic eye the enlargement is found by the following formula: The distance of the retina from the nodal point (optical center) of the eye is 15 mm. The observer projects the image which he sees to the point at which small objects are usually held, say 250 mm. The enlargement of the disk is proportional to these two distances, 15 : 250 : : 1.5 mm. : 25 mm. 16.6 = the enlargement. It is comparable to looking at the disk through the lens of 15 mm. focus, 66 diopters.

It is to be remembered that the farther this image is projected, the larger it appears. In hyperopia the enlargement is less than this. In myopia, on the contrary, it is greater.

Indirect Method.—In the indirect method of ophthalmoscopy a real, inverted image of the interior of the eye is obtained by means of a strong convex lens (object-lens), the principle involved being similar to that of a compound microscope.

The observer holds the object-lens (a convex lens of about 20 diopters) close to the patient's eye, and, placing a convex lens of 5 diopters (eye-piece) behind the ophthalmoscope, throws the light into the pupil and moves his eye nearer to or farther from the patient's eye until he distinctly sees a vessel or a portion of the nerve—that is, a real image of the eye-ground is formed by the object at its focal distance in front

of the eye. The observer sees this image, in which all the relations of objects are reversed. His eye is at a distance from the image equal to the focus of the lens in the ophthalmoscope—viz., 20 cm.

The image being inverted, the lower portion of it corresponds to the upper part of the eye-ground, and the right side of the image corresponds to the left side of the eye. If the observer moves upward, the image moves downward; if the observer moves to the right, the image moves to the left. Consequently, the upper part of the image must be viewed if it is desired to see the lower part of the eye-ground, and the right side of the image if parts of the fundus to the left are to be examined.

A comparison between the images as seen by the direct and indirect method may be stated thus: If, in the *direct method* with the disk in view, the observer moves his head to the right, he brings into view a

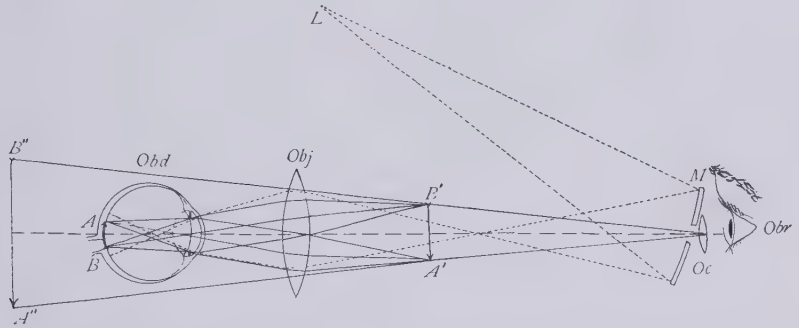


FIG. 51.—Diagram of the indirect method giving an inverted image: Rays from the source of light, *L*, converged toward the observed eye, *Obd*, by the concave mirror, *M*, are intercepted by the lens, *Obj*, and after coming to a focus diverge again and light up the fundus. From a part of the illuminated fundus *A–B* rays pass out of the pupil to be again intercepted by the lens *O* and form an inverted real image at its anterior focus *A′–B′*. This real image is viewed by the observer's eye behind the sight-hole of the mirror with the aid of a magnifying lens, *Oc*, and is seen enlarged, as at *A″–B″*. (B. A. Randall.)

portion of the retina to the left of the disk. The disk now moves out of the field toward the right, and disappears behind the right edge of the pupil. The image, therefore, moves with the observer. If, in the *indirect method* with the image of the disk in view, the observer also moves his head to the right, he sees the image of the same portion of the retina as in the direct method; but this being to the left of the disk, its image occupies a point to the right of that of the disk. The disk thus appears to have moved toward the left. The image, therefore, moves contrary to the movement of the observer's head. Movements in other directions are explained in the same way.

The *formation* of the *inverted image* in ophthalmoscopy may be understood by examining Fig. 51.

In hyperopia and emmetropia a convex lens is necessary to render the rays convergent. In myopia the rays emerge convergent, and the convex lens may be dispensed with in the higher grades, though it is

still an advantage because it increases the area of the fundus visible at one time.

Size of the Image.—The enlargement of the image in this method is less than it is in the direct method, but a larger portion of the eye-ground is visible at one time.

The size of the real image of the eye-ground of an emmetropic eye formed by the convex object-lens held at its own focal length from the eye is determined by the following formula: The size of the disk is to the size of the image as the distance from the retina to the nodal point (15 mm.) is to the focal length of the object-glass. If the lens has a focal length of 75 mm., the ratio is 15 : 75; the enlargement is then 5 diameters. A lens of 60 mm. focus would equal an enlargement of 4 diameters—15 : 60.

The observer will see this image under a higher angle in proportion as he comes closer; it will then appear larger. To do this he must either use his accommodation or place a convex lens (eye-piece) behind the ophthalmoscope. When the eye-piece is used, a visual image of the aërial image, still more enlarged, is produced, just as in the compound microscope. If the object-lens is withdrawn farther than its focal length from the observed eye, the image in myopia becomes larger, in hyperopia smaller, and in emmetropia remains the same. If the lens is brought closer to the eye, the image becomes smaller in myopia and larger in hyperopia.

Ophthalmoscopy.—The investigation of the deeper structures and interior of the eye by means of the ophthalmoscope may, therefore, be practised with (1) the direct, and (2) the indirect method.

1. **The Direct Method (Method of the Erect or Upright Image).**—The patient should be seated in a darkened room with his back to the source of illumination—an Argand burner or properly constructed and shaded electric light being suitable—which is placed behind and to the side of his head, on a level with the ear, the face being in shadow, while the rays of light just fall upon the outer canthus of the eye. This will enable the observer to come quite close to the eye without interfering with the path of the illuminating beam. The surgeon sits at that side of the patient which corresponds to the eye under examination—for example, the right—his position being preferably on a slightly higher level than that of the subject. He now takes the ophthalmoscope in his right hand, looks through the sight-hole with his right eye, at a distance of about 50 cm. from the observed eye (the convex border of the instrument being in contact with the concave margin of his brow), meanwhile keeping the other eye open, and reflects the light into the right eye of the patient. If the left eye is to be examined, the ophthalmoscope is held in the left hand.

If an *electric ophthalmoscope* is used, the instrument carries the source of illumination. Moreover, it is not necessary that the room shall be darkened.

If the patient looks directly into the light, the pupil, provided this is not dilated with a mydriatic, will contract and no satisfactory view

is possible. He must hence be directed to turn the head slightly to the right, and gaze into vacancy in the farthest limit of the room,

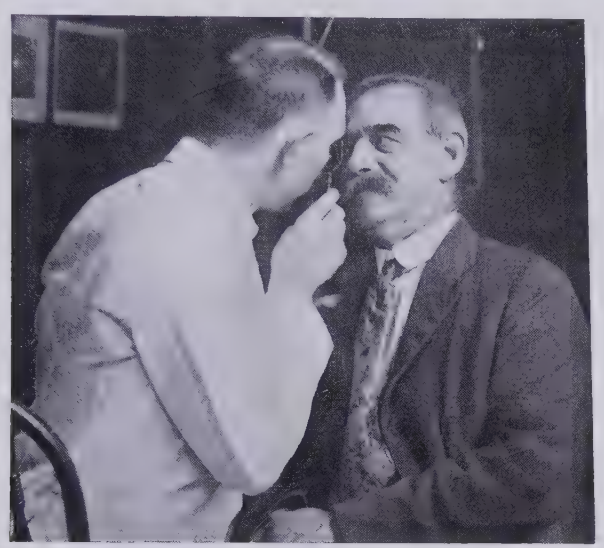


FIG. 52.—Ophthalmoscopic examination. Method of the upright image. Observer in the correct position.

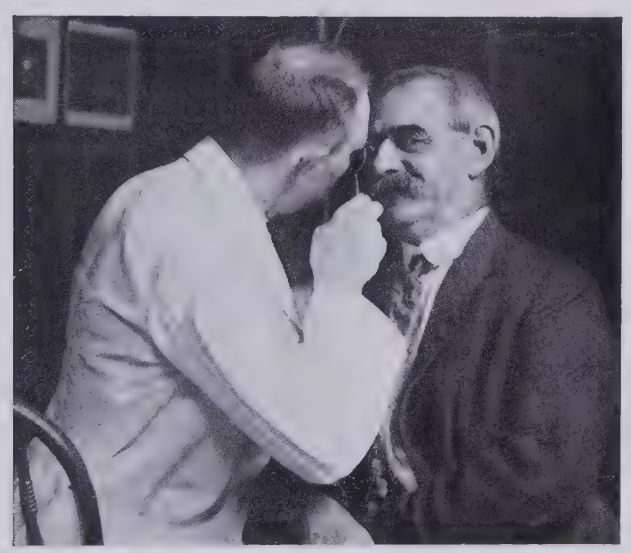


FIG. 53.—Ophthalmoscopic examination. Method of the upright image. Observer in an incorrect position.

when the pupil will be seen illuminated by a red glare—the reflection from the choroid coat—bright if the pupil is large, and dull if it is small. No details of the fundus are as yet visible at this distance (50

cm.) unless a certain grade of myopia is present or a considerable degree of hyperopia (see page 116).

The beginner should now practise keeping the light steadily in position, and may estimate the success of his endeavor by observing the glare in the pupil. If this changes in color or disappears, the light has shifted from its proper position because the examiner has failed to retain his elbow in close contact with his side, and allowed it to move outward and away from his body, the head meanwhile being bent to one or the other side of the vertical position it should assume in a direct line with that of the subject—feature to feature. This may be understood by observing the two accompanying illustrations (Figs. 52 and 53).

Having gained control of the light, the observer gradually approaches the eye of the patient, taking care that the red glare, which is tinted slightly yellow on the nasal side, marking the position of the optic papilla, remains unaltered, and comes as close as possible—within 1 inch or even nearer. If the maneuver has been successful, and the light directed slightly toward the nasal side, the most prominent feature in the eye-ground—the optic nerve—will come into view; or a retinal vessel may first be manifest, and should be followed to the papilla as a stream would be to its source.

Before proceeding to study the details of the fundus the student should make certain preliminary examinations.

(*a*) **Examination of the cornea, anterior chamber, and lens by transmitted light** is made by placing a + 7 D or + 16 D lens behind the mirror, and by coming close to the eye—that is, until the object to be examined is within the focal distance of the lens employed, and reflecting the light into the eye in the manner already described.

A foreign body on the cornea, a macula, a deposit on the posterior layer of the cornea, or an opacity in the lens appears as a black object against the red background, in contradistinction to its appearance in its true color under oblique illumination (see page 52).

At the same time the mobility of the iris should be tested, and an observation made as to whether the iris reacts promptly and evenly under the influence of the light directed into the pupil at various angles. Iritic precipitates in the anterior chamber and attachments to the lens may be observed by this method and by holding the ophthalmoscopic mirror practically at right angles to the eye the angle of the anterior chamber can be studied.

(*b*) **Examination of the vitreous** is made by reflecting the light with the concave or, better, the plane ophthalmoscopic mirror, from a distance of 30 cm. into the eye, while this is moved in various directions so as to bring into view opacities which have a lateral situation or which have sunk to the bottom of the vitreous chamber.

Vitreous opacities and detached retina are seen in the erect position if the observer is sufficiently far away, because they are within his range of accommodation. Small vitreous opacities appear dark; larger ones have a grayish appearance. If he approaches closely, he must

place behind the mirror a convex lens, in the manner just described, to bring them into focus, and should always use this method.

(*c*) **Location of Opacities in the Transparent Media.**—If, the observer using the ophthalmoscopic mirror in the manner described in the previous paragraph, an opacity is seen to be freely movable, it must be in the vitreous. Should the opacity move only with the movement of the eye, but not spontaneously, it probably is situated in the cornea or in the lens, although it may be present in the vitreous in the form of a fixed opacity. In these circumstances a differential diagnosis can frequently be made by means of oblique illumination. Should this method prove insufficient, the situation of the opacity may be ascertained by means of its parallactic movement in relation to the border of the pupil. Fuchs states the rule as follows:

The observer looks directly forward into the eye and notes the position of an opacity within the pupillary space. Next, while the patient keeps his eye entirely quiet, the examiner slowly moves from side to side and observes if the opacity retains or does not retain the same position in the pupillary space. If the opacity retains its position unchanged, it lies in the pupillary plane upon or immediately under the anterior capsule of the lens. If it does not retain its original position, it is situated in front of or behind this plane—in front of the plane if the opacity moves in a direction opposite to the direction of the movement of the observing eye, and behind the plane if the opacity moves in the same direction as the observing eye. The quicker the change of position takes place, the farther is the opacity removed from the pupillary plane.

Instead of proceeding in this manner, the observer may retain his position unaltered and cause the patient to move his eye in various directions.

When an opacity is far back in close relation with the retina, its location may be judged by noting its relation to the movement of the retinal vessels. How far forward it lies in the vitreous may be accurately measured by means of convex lenses (see page 117).

Having ascertained that the media are clear, and having approached sufficiently close, the details of the fundus oculi are brought into view and studied *seriatim*.

If either surgeon or patient is myopic, the necessary concave lens which corrects the error must first be placed in position; while, if hyperopia exists, the fundus is visible without the aid of a glass, provided the hyperopia is not in excess of the power of accommodation.

Beginners, however, often fail to obtain an image of sharp definition, owing to inability to relax accommodation, and succeed in seeing the details clearly only through a concave glass. The power of relaxing the accommodation comes with practice.

The **optic nerve** appears as a nearly round or slightly oval disk, situated toward the nasal side, varying in color from a grayish pink to a more decided red, the tint being most marked upon the nasal half, while the center is occupied by a whiter patch—the "light-spot"—

marking the position of the entrance and emergence of the retinal vessels. The general tint of the optic disk varies with the age and complexion of the patient and with the intensity of the color of the surrounding eye-ground.

The papilla is bounded by two rings. The outer one, dark colored, usually incomplete or existing only as a slight crescent of pigment upon one or the other side, is the *choroidal ring*, and represents the border of the choroidal coat, where this is pierced by the optic nerve. Within this is a faint white stripe, more distinct in elderly people, the *scleral ring*, which indicates the rim of the sclerotic coat, or, according to Loring, the connective-tissue elements of the inner sheath of the nerve (*connective-tissue ring*). The choroidal ring is often imperfectly produced or entirely absent, and the scleral ring may be faintly marked, visible only on one side of the disk, and in some eyes is scarcely if at all evident.

The central white patch may be noticeable only by contrasting it with the color of its surroundings, or it may be a distinct excavation, occupying the center of the disk, and having sharp borders, one of which often shelves slightly outward. This is the *physiologic cup*, and is the space left by the radiation of the nerve-fibers toward the retina, having a floor of white color, because it is composed of the interlacing opaque fibrous tissue, or *lamina cribrosa*, which underlies the optic papilla. It is often stippled in appearance, owing to the lack of light reflected by the non-medullated nerve-fibers, which pass through the spaces of the lamina. According to Schoen, the so-called physiologic excavations are due to dragging of the vaginal processes of the optic nerve and lamina cribrosa from overexertion of the accommodation, and hence are found in adult eyes more commonly than in the eyes of children. They are usually but not always bilateral, and one may be larger than the other. Schweigger traced hereditary transmission in some large physiologic excavations, but doubted if they were associated with any particular refractive condition of the eye.

The Blood-vessels.—From the central light-spot the *principal retinal arteries* emerge, and into it the *chief venous trunks* empty. Usually one venous and one arterial stem pass directly upward and downward, and on the edge of the disk, or a short distance from it, each divides into two branches. Sometimes this division has taken place in the axis of the nerve behind the lamina, and two arteries and two veins appear directly in the central opening of the papilla, or *porus opticus*. The arteries traverse the surface of the eye-ground, dividing dichotomously into numerous branches, and, passing above and below, spread in greater size and number over the temporal half of the retina, sending small branches toward the macula; and in smaller size and less number over the nasal side. Fine branches arising from the central large trunks, or springing directly from the nerve, pass outward and inward, and also undergo numerous divisions.

The veins pass over the eye-ground in the same general direction as the arteries, and in close relation to them, emptying usually by means of two large branches into the center of the disk.

PLATE I.



The normal fundus of the right eye examined by the direct method of ophthalmoscopy.

According to the situation of the vessels, they are named, respectively, upper and lower temporal artery and vein, upper and lower nasal artery and vein, and macular and nasal arteries and veins.

The veins are dark red in color, contrasting with the bright, natural, blood-red color of the arteries. They are slightly tortuous, and larger than the arteries in the proportion of 3 to 2. The difference in color between veins and arteries is most marked in the major branches. In the finer twigs, after four or five divisions, the distinction between arteries and veins is often possible only by tracing them to their source.

Each vessel usually presents a double contour, owing to a bright stripe which passes along the center, leaving a red line on either side. This so-called *light-reflex* has been ascribed to a condensation by the refractive action of the blood column of the rays of light which have passed through the vessel from in front, and have been reflected back slightly from the posterior wall, but chiefly from the underlying tissues.[1] It is more marked upon the arteries than upon the veins, and, indeed, is often absent as the latter cross the disk, being visible in a minor degree when they lie at some distance in the retina.

Pulsation.—The retinal arterial pulse, in general terms, due to varying pressure of the blood column, has been classified by Ballantyne as follows: (1) *Locomotor pulse*, that is, a rhythmic displacement of the artery almost synchronous with the cardiac systole; (2) the *expansile pulse*, being a broadening of the blood column; (3) the *capillary pulse*, being a variation in the tint of the optic disk, reddening occurring during systole and paling during diastole, and (4) the *pressure pulse*, a rhythmic collapse or disappearance of the artery, observed in glaucoma, and produced by pressure on the eyeball, and sometimes called the *collapsing pulse*. The locomotor pulse is physiologic, at least not pathologic; the expansile pulse may occur in healthy persons, or may be associated with aortic insufficiency; the capillary pulse is seen with aortic regurgitation; the pressure or collapsing pulse is observed in glaucoma, in syncope, in orbital tumors, and in optic neuritis. A true pulse wave and one which may extend beyond the disk may be in association with aortic regurgitation, aortic aneurysm, and exophthalmic goiter (see also page 670).

Spontaneous pulsation in the veins of normal eyes is a frequent phenomenon and is especially noticeable at or near the edge of the disk. Lang and Barrett found it in 73.8 per cent.; Veasey, in 58.3, and the author, in 62.1 per cent. of their examinations. Parsons points out that the venous pulse is diastolic, and may, therefore, be called a *negative venous pulse;* in pathologic circumstances, for example, in tricuspid re-

[1] The cause of the light streak was usually attributed to reflection from the anterior surface of the vessel wall or the anterior surface of the blood column, until Loring maintained that the *refraction of light* was the chief cause of the phenomenon. A. E. Davis endeavored experimentally to confirm Loring's conclusion. Story rejects Loring's theory, and assumes that the reflex comes from the vessel walls. Dimmer ascribes the retinal reflex to the axial blood-stream in the arteries and in the veins to the surface of the blood-stream. Elschnig believes that both in arteries and veins it is the surface of the blood column which causes the reflex.

gurgitation, it is presystolic and is called the *positive venous pulse*. It may be produced by a slight pressure upon the globe. The spontaneous pulse is due to a communication of the arterial pulsation to the vein, as these vessels lie side by side in the optic nerve, or may be explained by the theory of Donders, that during the systole of the heart (diastole of the retinal arteries) an increased tension in the vitreous is communicated to the walls of the retinal veins, especially the larger ones, at their exit from the eye where the least resistance is offered, obstructing the flow of blood and compressing their lumen. The blood coming from the capillaries overcomes this resistance and the vessels regain their caliber, alternate collapse and distention thus being produced. According to Türk, the venous pulse is due to a continuation of the arterial pulse-wave through the capillaries into the veins.

The blood-pressure in the vessels of the retina has recently been elaborately studied by Bailliart and Magitot with the aid of a special *dynamometer* designed by the former observer. According to their researches the arterial pressure is always higher than the ocular tension, but the venous pressure is not always as great as the ocular tension; pulsations occur when the pressure exerted by the ocular tension on the wall of the vessel balances the pressure of the blood column.[1]

Physiologic Variations.—The *papilla*, instead of being round or slightly oval, with a vertical long axis, is often distinctly irregular in outline, or has its long axis in a horizontal or oblique direction. Its outer half may be embraced by a crescent of greater or less choroidal changes, the so-called *conus* or *crescent*. A congenital crescent of white appearance, the *underlying conus*, may sometimes be seen below (see page 541), and occasionally is very broad, approximating in appearance a coloboma of the nerve-sheath (see page 540).

The *physiologic cup* varies in size, area, and depth. Normally situated on the temporal side, it may be a deep pit, funnel shaped, with overhanging margins over which the vessels sharply bend, or very shallow and dish-like, sloping to the temporal side, or deep and sharply marked on its inner side, but shading outward.

The *distribution of the vessels* is subject to numerous variations—so much so that it would be difficult to find it the same in any two eyes. The usual departure from the ordinary type is the one already referred to, in which four major branches (two arteries and two veins) appear at the center of the porus, instead of two large branches which later divide at or near the margin of the disk. Anomalies of the veins upon the disk, in the form of unusual bifurcations, are occasionally seen. Division of the vein just before entering the disk; division at the margin; the formation of a vascular circle and final reunion in a single vessel; and anastomosis of the central vein with an aberrant vein, or one which has penetrated the inner side of the disk, have been described (Randall). The veins are normally more tortuous than the arteries. Both sets of

[1] For a further consideration of this subject consult American Journal of Ophthalmology, 1922, v, 777, and Trans. International Congress of Ophthalmology, 1922, 348, Washington, D. C. La Circulation Rétinienne, par Bailliart, Paris, 1923.

vessels present this appearance in marked degree in certain pathologic conditions, but also occasionally as an anomaly without such significance (Fig. 54). Again, the vessels may stand forward from the disk in a high curve, or twine around each other, as we sometimes see two stems on a vine. Marked enlargement of the anastomoses about the nerve entrance, which anastomoses, according to Leber, connect the retinal and ciliary vascular systems at the level of the papilla and at the level of the choroid, have been described by George Coats.

An anomaly of not infrequent occurrence (7 to 10 per cent. of examined eyes) is a *cilioretinal vessel*, usually, according to Elschnig, an artery, which appears at the temporal border of the disk, then arches

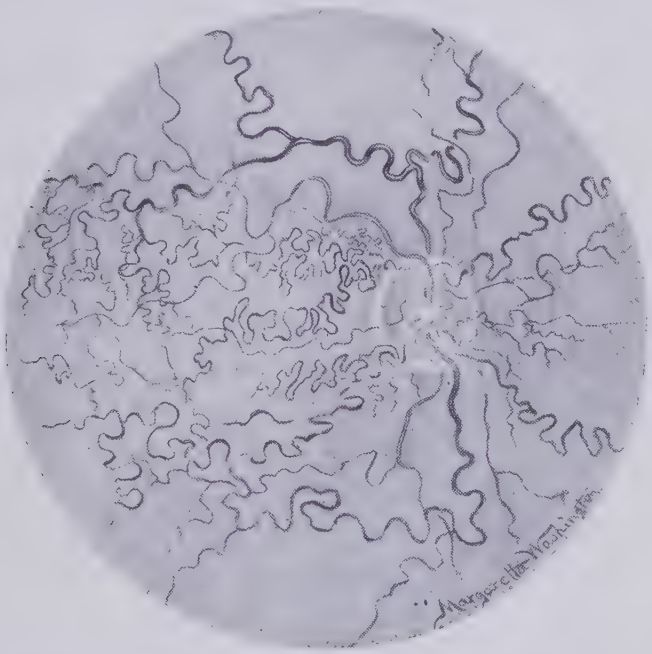


Fig. 54.—Extreme congenital tortuosity of vessels. Note the breadth of the scleral ring.

outward or away from the papilla, enters the retina, and pursues a general course toward the macula. A large cilioretinal vessel may take the place of one of the temporal arteries. According to Elschnig, a cilioretinal vessel may be a primary branch of a ciliary artery which pierces the sclera obliquely, without sending a branch to the choroid, and then enters the intrascleral or intrachoroidal part of the optic nerve, or an offset of a ciliary vessel which primarily enters the choroid, where it divides, and one branch passes on into the retina and produces the anomaly in question. Cilioretinal veins may arise from the choroidal vascular system. *Opticociliary vessels* are uncommon. They are practically always veins, only two instances of opticociliary arteries having been reported (Coats). They pass from the central vessels to the disk

border, where they disappear under the retina into the choroid. In reporting a case of this character W. T. Shoemaker regards the anomalous vessel as representing an *aberrant choroidal vein.*

The Retina. —Inasmuch as the retina is practically transparent, a study of this membrane is hardly possible without a consideration of its underlying pigment epithelium of the choroid and even of the sclera.

In certain persons, especially of dark complexion, the retina assumes a grayish tint in the neighborhood of the papilla, most marked at its nasal half. This faint opacity is slightly streaked, the striations indirectly corresponding to the expansion of the optic nerve-fibers. Eyes long subjected to the strain of uncorrected ametropia furnish an exaggerated picture of this appearance, which, if at all extensive and associated with similar opacities along the lines of the vessels, assumes pathologic importance (see Retinitis). In old people the retina is less transparent than in those of younger years; the vessels are often smaller and the nerve-head paler.

In the eye-ground of young subjects, particularly along the line of the vessels, numerous wave-like, glistening reflexes may be seen to follow one after another with the slightest movements of the ophthalmoscopic mirror. The effect is similar to the shimmer seen on the surface of certain silks, and has been designated by English writers *shot-silk retina.* It is unusual to find the phenomenon in individuals over thirty, its occurrence being marked in direct proportion to the youth of the subject. This appearance is without pathologic significance.

Macula Lutea.—About two disks' diameter to the outer side of the papilla, and slightly below the horizontal meridian, there is a circular or slightly oval spot, equal in area to the end of the optic nerve, darker in color than the surrounding fundus, uncrossed by any visible retinal vessel, but toward which the finer twigs of the major branches pass, fringing its boundary. This region is the *macula lutea*, or *yellow spot*, and is that portion of the eye-ground concerned with the functions of direct vision.

Its center is occupied by the *foveal reflex*, which marks the edge of the *fovea centralis*, and which may appear as a spot of light, a small circle with reddish center, a shifting crescent, or a shining line. This, in turn, is surrounded by a dark area (*the dark spot of the macula*), sometimes containing a number of brownish-black or light colored or even glistening granules, which have been mistaken for, and described as, Gunn's dots.[1] They have no pathologic significance. Finally, the margin of the macula is bounded by a glistening *whitish ring* or *halo* (macular reflex).

[1] Gunn's dots ("Crick" dots) were thus described by Marcus Gunn: "Very minute yellowish-white shining dots for some distance around the disk, especially to the nasal side and below. In distribution these dots are remarkably equidistant from each other and are situated anteriorly to the largest retinal blood-vessels, each being less than one-fifth of the diameter of a large vessel; the outline of the disk is rather indistinct, the large veins full and somewhat tortuous." Those first described occurred in the eyes of members of the same family. Their nature is unknown. Dread of light may be a conspicuous symptom.

The method of examination determines whether all these characteristics of the macula lutea can be observed. They are fairly constant, however, with the exception of the *halo*, and are notable in young children. Ordinarily, the macular ring is best seen in the inverted image in young eyes, where it is apt to assume an oval shape, that is, a delicate white curved line forms a horizontal oval, approximately the size of the papilla, which encloses a brownish-red area containing a bright dot in its center. According to Lindsay Johnson, even in the upright image, if the source of illumination be gradually lowered, a time is arrived at when more light is reflected from the macula than from the general fundus, and at that moment the ring appears. In elderly people the region usually cannot be well recognized except by the absence of vessels and its darker color, but even in them careful focusing will not infrequently reveal the foveal reflex. In albinos it is still more difficult to define this area.

Although no vessels visible to the ophthalmoscope cross the macula, except as an anomaly (Randall, Johnson), the region is abundantly supplied with capillaries, which can be shown by artificial injection, which surround the fovea in a close loop, but do not occupy it. The student may find the region difficult to study because the light falling upon it causes the pupil to contract, the view being further hindered by the corneal reflex. Hence the pupil should be dilated, when the macula may be brought into view by requiring the patient to look directly into the ophthalmoscopic mirror, or may be found by turning the light outward from the lower edge of the disk. The region should always be studied with the utmost care. The details in the macular region may be studied with advantage if a diaphragm lamp is used as the source of light for ophthalmoscopy by the direct method (*simple centric ophthalmoscopy*). The mirror used should be concave, with a rather short radius of curvature, for example, the small mirror of a Morton ophthalmoscope (Gullstrand).[1]

The appearances in the macula depend partly upon the disposition of the layers of the retina in this region. At its margin the retina is much increased in thickness by an extra development of the layer of the ganglion cells, while the fovea is produced by the hollowing out of the center of the macular region. The macular reflex, or ring, therefore, may be considered as a reflection arising from the thickened macular circumference, and the foveal reflex as a reflection from the edge of the fovea. The variations, according to Johnson, are due to the direction and the shape of the sloping sides of the pit, but, according to Dimmer, depend upon the kind of ophthalmoscopic mirror which is employed, the reflex being the inverted image of the center of the mirror. According to Piersol, the color of the macula depends upon the presence of a yellowish pigment within the layers internal to the visual cells, the latter elements remaining colorless; in consequence of this arrangement

[1] For the technic of Simple Centric Ophthalmoscopy and Centric Reflexless Ophthalmoscopy, see Gullstrand, Transactions of an International Congress of Ophthalmology, Washington, 1922.

the fovea, in which the neuro-epithelium alone exists, is devoid of pigment and, therefore, appears as a light spot within the colored area. The dark brown spot of the macula is generally believed to depend upon the thinning of the retina at this spot, with a more decided pigmentation in the epithelium. Dimmer, however, thinks that it is also produced by absence of the slight veiling of the retina at this point, which is manifest in the surrounding more compact layers.

The Choroid.—The bright glare which illuminates the pupil when the light is thrown into it from the ophthalmoscopic mirror, and develops into the uniform red color of the fundus, when this is brought into view, arises from the choroid. The rays of light pass through the transparent retina to its pigment epithelium, which in ophthalmoscopic work is usually accredited to the choroid, and in part are absorbed and

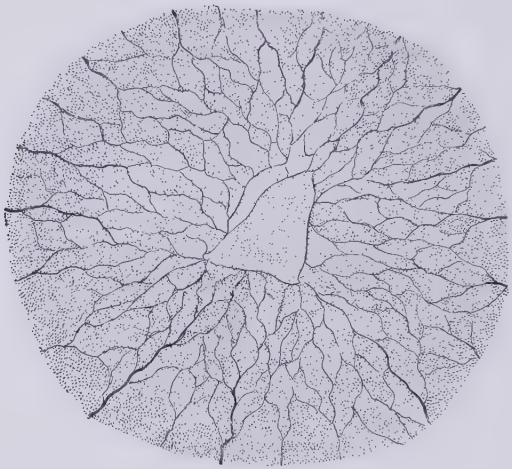


FIG. 55.—Minute vascularization of the macular region as shown by entoscopic study of the right eye illuminated through a moving pin-hole. (Randall, American Text Book of Diseases of Eye, Ear, Nose, and Throat.)

in part reflected. The greater the quantity of the pigment, the greater the amount of absorption, so that the color of the eye-ground depends upon the degree of saturation in this epithelium, and varies from an almost slaty color in the dark-skinned races to a dark red in persons of blond complexion. A light yellowish-red or brownish color is often evident.

In very fair people the diminished amount of pigment contained in the pigment epithelium and the imperfect development of pigment-cells of the choroid expose the larger choroid vessels, which are evident as a meshwork of tortuous red bands with intervening spaces of lighter or darker color (intervascular spaces), and which are distinguishable from the retinal arteries and veins by their flat appearance and absence of the light streak. An eye-ground of this appearance is known as the *tessellated fundus;* it must not be mistaken for choroiditis (page 387).

A nearly perfect exposure of the choroidal vessels is seen in albinos (*albinotic fundus*). The intervascular spaces are lighter than the vessels because the sclera is visible.

It is not usually possible with the ophthalmoscope to differentiate the arteries and veins of the choroidal vascular system, although the latter are of greater size, and, near the equator of the eye, converge toward the venæ vorticosæ, being separated by larger and longer spaces. In decided brunettes these spaces are more deeply tinted than the vessels, which appear "like light streams separated by dark islands" (Nettleship). A fair general idea of what tint may be expected in the fundus may be obtained by observing the color of the patient's hair.

All the details of the eye-ground may be studied with greater ease through a dilated pupil, and, on beginning his studies, the student may with propriety employ a mydriatic—euphthalmin, cocain, or homatropin, not atropin—provided no signs of glaucoma are present and at the conclusion of the examination a drop of $\frac{1}{2}$ per cent. solution of pilocarpin is instilled. Having acquired a knowledge of the normal appearance thus seen, he must now practise with the undilated pupil.

The disk and macula having been studied, the peripheral parts of the eye-ground should be examined by throwing the light inward, upward, and downward, the head of the observer being moved correspondingly to comply with the changed direction of the mirror. Even where the central part of the fundus presents the usual characteristic red tint, the choroidal vessels are frequently exposed in the periphery, presenting the appearance just described, and having no clinical importance.

Ophthalmoscopy with Red-free Light.—That changes in the color of the eye-ground are produced by variations in the color of the source of the illumination employed in the examination is well known. Thus Mayou, working with a mercury vapor lamp, noted that the general color of the fundus was green, the optic disk white in its center and green at its edges, the retinal vessels purple, and the choroidal vessels a deeper purple.

In comparatively recent times, within the last ten years, the examination of the fundus by means of light from which the red rays have been excluded by a suitable filter has attracted much attention and study. We are particularly indebted to A. Vogt for researches in this respect. The source of the light may be an arc lamp equipped with a suitable aqueous filtering solution, or a glass screen. Recently electric ophthalmoscopes, so arranged that a glass filter may be slipped into place, have been constructed, which apparently are destined to serve a useful purpose, and whereby more cumbersome apparatus becomes unnecessary. To be effective the light which is thus filtered must be exceedingly bright.

In general terms, using red-free light, the choroid becomes less visible and the retina more visible. The normal fundus presents a greenish-yellow tint; the optic disk is greenish white; the macula is yellow, the

yellow area being about one-third the size of the papilla; the vessels appear dark, the smaller ones black; and the retinal reflexes are markedly visible at all ages. A striking feature is afforded by the radiating retinal fibers. Certain pathologic changes are readily detected; small retinal hemorrhages which appear dark, perivascular striations and fold formations around the macula. Observations should be made through a dilated pupil, if possible, preceded by the usual ophthalmoscopic examination, in order that a comparison between the two methods may be made.

Determination of Refraction by the Ophthalmoscope.—The estimation of the refraction of the eye by means of the ophthalmoscope results in either a *qualitative* or a *quantitative* determination.

The former is obtained in the following manner: Hold the ophthalmoscope 30 to 50 cm. from the patient's eye, and, looking through the central aperture of the mirror, unaided by a glass, observe if any vessels come into view. Their appearance means that the eye is either hyperopic or myopic. Now move the head from side to side, and note if the vessels move apparently in the same or in a direction opposite to the movements of the head. If the former, the eye is hyperopic; if the latter, myopic. Inasmuch as the image of the vessels in low degrees of myopia would be formed only at a considerable distance from the observed eye (30 to 120 cm.), and since no sharp image would be obtained in either emmetropia or low degrees of hyperopia farther away than 30 cm., any considerable degree of ametropia may be excluded by failure to obtain a direct image except at a long range or a very short distance from the patient's eye.

In attempting a *quantitative* estimation of the refraction of the eye it should be remembered that the strongest convex lens, placed in position in the ophthalmoscope, with which the emmetropic observer can still see the details of the fundus at the point selected, indicates the degree of hyperopia; the weakest concave lens, of myopia. The hyperopia usually is somewhat greater, and the myopia somewhat less, than the result obtained by ophthalmoscopic examination.

In order to estimate the refraction of the eye examined the hyperopic observer must subtract from the convex or add to the concave lens, which yields him a sharp image of the fundus, the amount of his own error; while the myopic observer must add to the convex or subtract from the concave lens, with which he sees the eye-ground, the degree of his own near-sightedness.

In order to calculate the amount of lengthening or shortening of the eye equal to a lens which neutralizes the myopia or hyperopia in any given case, and provided the distance between the surgeon's eye and that of the patient is not more than 2.5 cm., the following table, which was prepared by the late Mr. Nettleship, is useful:

Hyperopia of	1 D	represents a shortening of	.....	0.3	mm.
"	2 D	" "	.....	0.5	"
"	3 D	" "	.....	1	"
"	5 D	" "	.....	1.5	"
"	6 D	" "	.....	2	"
"	9 D	" "	.....	3	"
"	12 D	" "	.....	4	"
"	18 D	" "	.....	6	"
Myopia of	1 D	represents a lengthening of	.....	0.3	"
"	2 D	" "	.....	0.5	"
"	3 D	" "	.....	0.9	"
"	5 D	" "	.....	1.3	"
"	6 D	" "	.....	1.75	"
"	9 D	" "	.....	2.6	"
"	12 D	" "	.....	3.5	"
"	18 D	" "	.....	5	"

By this table the depth of an excavation in the papilla may be measured. For instance, if the bottom of the pit required − 5 D for its sharp examination, and the margin of the nerve was seen without any glass, the depth of the excavation would be 1.3 mm.

The presence of *astigmatism* may be ascertained by means of the ophthalmoscope and the upright image because all points of the portion of the fundus under examination are not in focus at the same time—*e. g.*, the retinal vessels running in the directions which correspond to the principal meridians.

Thus, when two vessels cross each other at right angles, the vertical branch may be sharply seen, while the horizontal one presents a blurred image, or the upper and lower margins of the disk may be clear, but the lateral borders indistinct. The amount of hyperopia or myopia of the *vertical* meridian is equal to the strongest convex, or weakest concave, glass which makes distinct the vessels running in a *horizontal* direction. The refraction of the *horizontal* meridian is determined by the glass which yields a clear image of the vessels running in a *vertical* direction. As the vessels do not correspond to the layer of the rods and cones, the measurement is an approximation.

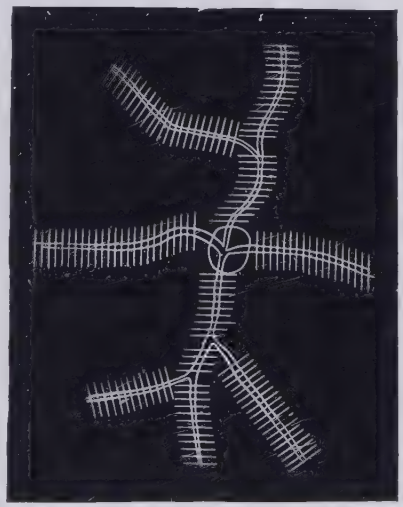


Fig. 56.—Focusing of the vessels by the meridians of an astigmatic eye; the parallel lines on each vessel represent the direction of the meridians through which a distinct image of the vessel is obtained.

Compound astigmatism is determined by finding, in hyperopia, the strongest convex lens which the vessels in each meridian will bear with the preservation of a distinct image, and subtracting the one from the

other, thus finding the difference between the meridians—*i. e.*, the amount of astigmatism. In high degrees of astigmatism the optic disk usually appears as an ellipse, its long axis corresponding with the meridian of greatest refraction.

The measurement of astigmatism in this manner, with any degree of accuracy, requires much practice, a perfect control of the accommodation, and even then must never be employed to the exclusion of other and more trustworthy methods.

2. **The Indirect Method (Method of the Inverted Image).**—The patient and surgeon are seated in the same relative positions as have already been described in connection with the direct method, and, if the right eye is to be examined, the ophthalmoscope is held in the right hand at a distance of 30 cm. from the patient, who is instructed to look at the right ear of the examiner. A convex lens of 20 D, held

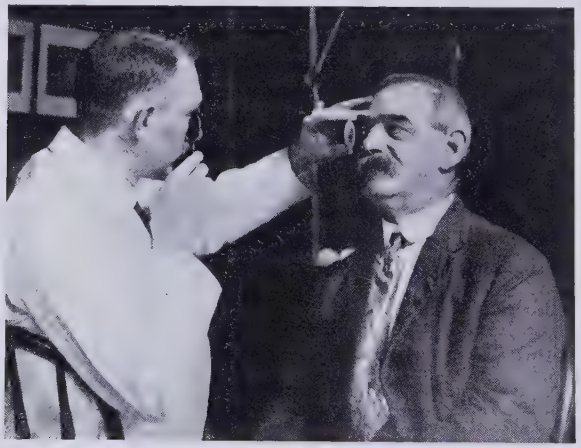


FIG. 57.—Method of an indirect examination with the ophthalmoscope.

between the surgeon's left thumb and index-finger, while the remaining fingers are rested upon the brow to steady the hand, is placed at about its own focal length in front of the patient's eye, directly in the path of the rays returning from the fundus, which are thus brought to a focus and form an aërial image between the observer and the glass.

If the left eye is to be examined, the ophthalmoscope is held in the left hand, and the patient instructed to look at the surgeon's left ear, while the lens, grasped in the fingers of the right hand in the manner just described, is placed in position.

The image which is found at a certain distance in front of the object-glass may not present itself to the observer as a distinct picture, owing to his inability to accommodate for the point of its formation. This accommodative strain may be relieved and the image magnified by placing behind the ophthalmoscope a convex glass of 5 D, which adapts the emmetropic observer, with relaxed accommodation, for a point 20

cm. distant. If the observer is presbyopic, or has a deficient amplitude of accommodation, this additional lens is absolutely necessary; while if he is hyperopic, the degree of his hyperopia should be added to the glass used as a magnifier. The observer possessing a moderate degree of myopia requires no lens in the ophthalmoscope, because he views the aërial image at his far point, while if his myopia is of high grade, he will need a weak concave glass.

With the indirect method of examination the field is larger than in the direct method. Although individual objects in the field are small and sharply defined, the details of the fundus are less perfectly revealed than with the direct method. In younger subjects in the macular region a bright reflex encircles an elliptic dark area containing in its center a reddish or, less frequently, a bright point surrounded by a small brilliant ring. These characteristics are not always present.

A *qualitative* estimation of the refraction with the indirect method may be made with the mirror alone, in the manner already described (see page 116). Furthermore, ametropia of high degree may be recognized by varying the distance of the object-lens from the eye. Withdrawal of the lens from the eye causes the image to appear smaller in hyperopia, larger in myopia.

The measurement of the degree or quantity of the refraction and the estimation of astigmatism by the indirect method were at one time much practised, but in practical work the methods are so far inferior to those usually employed (skiascopy, ophthalmometry) that their description is omitted.

Ophthalmodiaphanoscopy.—With the ophthalmodiaphanoscope, designed by Carl Hertzell, it is possible to make examinations of all parts of the eyeball and of the orbit around and posterior to the globe. Dr. H. F. Hansell thus describes the instrument:

> It consists of an 80-candlepower electric lamp, strengthened by a reflector, which increases the power of the light to 100 candlepower. The lamp is held by the patient far back in his mouth and a black mask is adjusted to protect the face. The anterior rays pass through the buccal plate of the superior maxillary bone, through the antrum and its roof to the floor of the orbit; the posterior rays pass through the hard and soft palate, the lateral walls of the nose, the anterior cells of the sphenoid, and pass to the median orbital walls. The eye-ground is illuminated from behind, below, and from the median side. The observer approaches the lighted pupil as near as possible and examines the illuminated fundus without the aid of any other instrument. If the eyes are ametropic (myopic) a correcting glass should be worn, and Hansell finds mydriasis advantageous.

According to Hertzell, the optic nerve appears more opaque than it does in ordinary ophthalmoscopic examination, its outlines are sharply defined, and if a pigment ring exists, it is distinct. The retinal veins are very dark, the arteries somewhat lighter in color; the macula appears as a dark spot. Retinal hemorrhages appear as dark, sharply defined areas. A tumor would diminish the illumination and obscure the fundus details. The brilliant transillumination of the sinuses and orbit is instantly obstructed by the presence of a growth, thickening, or opacity,

and the diagnosis thereby facilitated. The author's experience with the instrument is limited to a few observations made with Dr. Hansell; it seems to be of value in the diagnosis of orbital and sinus disease.

Ophthalmometry.—This term indicates mensuration of the eye, and, as usually employed, is limited in its application to the measurement of the radius of curvature of the cornea (*keratometry*). In order to practise ophthalmometry, instruments for taking the measurement of the radius of curvature of the cornea have been constructed, and are known as *ophthalmometers*. The ophthalmometer most in use is the one devised by Javal and Schiötz.

Other instruments are those designed by Leroy and Dubois, Reid, Hardy, Chambers-Inskeep, and Sutcliffe. In the opinion of the author, a suitable ophthalmometer, or, more accurately, keratometer, is of the greatest service in determining the refraction of the cornea and the direction of its principal meridians. None of these instruments should be used to the exclusion of other methods, especially the employment of mydriatics and skiascopy. (For a full description of the method of using the ophthalmometer see Appendix, page 798.)

Optometry is a term which indicates the principles involved in the measurements of the refraction of an eye by its limits of distinct vision. The instrument which thus serves to determine the refraction of the eye is called an *optometer*.

Optometers are based upon a number of principles. For instance, a single convex lens by which the direction of the luminous rays emanating from an object is changed, and consequently the determination of the refraction of the eye rendered possible, constitutes an optometer. Other optometers are based upon the principle of a telescope; still others upon the measurement of circles of diffusion, upon Scheiner's experiment, and upon the chromatic aberration of the eye. It would not be possible, in the limits of this manual, to describe in detail the principles involved or the various forms of apparatus which have been employed. Should the student desire to pursue the subject, he may with advantage consult the chapter devoted to this method found in Landolt's *Refraction and Accommodation of the Eye*.

Of the many instruments constructed for the purpose of estimating the refraction of the eye, and to which the name *refractometer* is usually applied, one of the best is the one devised by the late Dr. William Thomson.[1]

Skiascopy, or the Shadow-test (Retinoscopy)

BY EDWARD JACKSON, M. D.

This is a method of determining the refraction of the eye by watching how the light passes across the pupil when it is moved back and forth across the face by rotating a mirror which reflects it to the eye.

With the ophthalmoscope the observer, looking into a myopic eye from close in front of it, can see an erect image of the fundus, which he can render clear by the proper concave lens. In the same eye, from a

[1] Transactions of the American Ophthalmological Society, 1902, vol. ix.

greater distance, he can view an inverted image of the fundus. The point at which the change from the erect to the inverted image occurs is called the *point of reversal*. It is the focus conjugate to the retina, the point for which the eye is focused, the "far point" of distinct vision. Skiascopy is an accurate method of determining this point of reversal.

To apply the test with the *plane mirror*, the surgeon faces the patient at a distance of about 1 meter or less; holds the mirror before his own eye, and reflects on the patient's face the light from a lamp (the Shahan lamp is about the best) covered with an opaque shade with an aperture 2 or 3 mm. in diameter. By rotating the mirror the area of light it throws on the face is made to move up and down, or from side to side, or obliquely. The part of the light that falls on the patient's pupil is condensed on his retina, forming there a small light area which also moves as the mirror is rotated; for the plane mirror this retinal light area always moves in the same direction as, or "with," the light on the face.

In Fig. 58 *L* represents the source of light, screened from the patient, and *A* and *B* two positions of the plane mirror. When the mirror is at *A*, the light that enters the eye will come as though from a flame at *l*, to be condensed toward *a*, on the lower part of the retina. At this time



FIG. 58.—Skiascopy with the plane mirror.

the light falls on the lower part of the face. But when the mirror is rotated to *B*, the light entering the eye comes from the direction *l'*, and is condensed toward *b*, on the upper part of the retina. At the same time the light on the face moves upward. The positions of the retina in hyperopia, emmetropia, and myopia are shown at *H*, *E*, and *M*. In all forms of ametropia the real movement of the light on the retina is *with* the light on the face.

When skiascopy is practised with a *concave mirror* the source of light must be placed behind the patient; and the light area on the retina really moves in an opposite direction—"against" the light on the face, "against" the movement of the mirror.

In Fig. 59 the action of the concave mirror is represented. When the mirror is at *A*, the light that enters the eye diverges from the focus of the mirror at *l*, conjugate to the position of the light source, and is condensed toward *a*, on the upper part of the retina; and when the mirror is at *B* the light enters from *l'*, the new position of this conjugate focus, to be condensed toward *b*, on the lower part of the retina—that is, as the light has moved upward on the face, it has moved downward on the retina, and this is true for either *H*, *E*, or *M*.

The following account assumes the use of the plane mirror, but will apply equally to the concave mirror, if one bears in mind that with the latter the movement in the pupil is always in the opposite direction, and that the lens before the patient's eye must be changed, instead of changing the surgeon's distance from the patient.

We have described above the *real* movement of the light on the retina, as it would appear in the back of an enucleated eye with the sclera and choroid removed. But the surgeon does not see it in that way. He can only watch the *apparent* movement as seen through the pupil. This

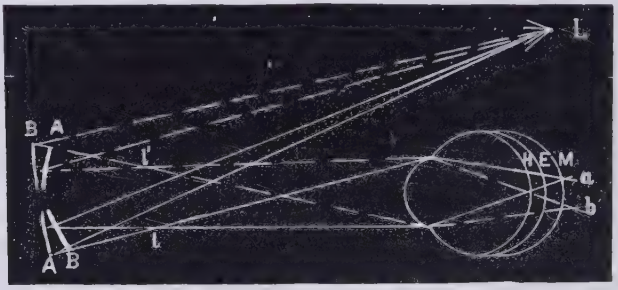


Fig. 59.—Skiascopy with the concave mirror.

will be (plane mirror) the same as the real movement, *with* the light on the face when he sees an *erect image*, and *against* the light on the face when he sees an inverted image.

In Fig. 60 *M* represents a myopic eyeball, from the retina of which rays come out and are focused at *B*, the *point of reversal.* Anywhere closer to the eye than this, as at *A*, an erect image is seen—the light in the pupil seems to move *with* the light on the face. Anywhere beyond the point of reversal, as at *C*, an inverted image will be seen, and the



Fig. 60.—Rays coming from a myopic eyeball.

light in the pupil will appear to move *against* the light on the face. Just at the point of reversal *B* it is impossible to see which way the light moves, and the illumination of the pupil is very feeble.

At one or two diopters from the point of reversal the light is comparatively bright. As the examiner goes farther than this from the point of reversal, it becomes more and more feeble. With the same movement of the mirror the apparent movement of the light in the pupil is quicker as the point of reversal is approached, and slower as it is departed from. These variations in the degree of illumination and ra-

pidity of movement may aid the expert in choosing the lens to be next placed before the eye, but the thing mainly depended on is the direction of the movement.

Application in Myopia.—If the surgeon, on throwing the light into the eye, finds that its apparent movement in the pupil is *against* the light on the face, he must be farther from the eye than the point of reversal (*B*, Fig. 60). He should then slowly approach the patient, still rotating the mirror and watching the apparent movement of the light until he finds this apparent movement is *with* the light on the face, as at *A*. He is now closer to the patient than the point of reversal, and should draw back and observe the greatest distance (*A*) at which this movement with the light on the face can be distinguished; then, drawing farther back, he observes the nearest point to the eye (*C*) at which the inverted movement can be seen, and the point *B*, half-way between *A* and *C*, is to be taken as the point of reversal. These observations should be repeated until the exact position of *B* is established. The distance from *B* to the eye is then measured; it is the focal distance of the glass required to correct the myopia. For instance, if the erect movement is seen as far as 55 cm. from the eye, and the reversed movement as near as 80 cm., the point of reversal will be about 67 cm. ($\frac{2}{3}$ of a meter) and the myopia, therefore, 1.50 D. ($\frac{3}{2}$ of 1 diopter).

If the myopia thus discovered is high, its amount can be most accurately determined by putting on a concave lens, that will correct all of it but 1 or 2 D, measuring what is left; and adding this to the strength of the lens used, to get the total myopia.

If, on the other hand, the myopia is very low, the point of reversal may be at so great a distance that from it one cannot see which way the light is moving in the pupil. In this case a weak convex lens must be placed before the eye, the point of reversal found with the lens, and then the strength of the lens deducted from the myopia which this indicates, in order to find the myopia of the eye.



FIG. 61.—Rays emerging from a hyperopic eye.

Application in Hyperopia.—Here the rays from the retina emerge divergent, as shown by the broken lines in Fig. 61, the eye has a virtual far point behind it and there can be no point of reversal in front of the eye. The surgeon finds the apparent movement of the light in the pupil is *with* the light on the face, and it continues to be so, no matter how far he draws back. It is necessary, then, to place a convex lens (*L*) before the eye, strong enough to render the rays convergent, and so to make a point of reversal (*B*) a convenient distance in front of the eye.

This lens does two things: First, it overcomes the divergence of the rays; this takes part of its power. Second, the remainder of its power makes the rays converge, causing a sort of artificial myopia. The point of reversal (*B*) obtained is the point of reversal for this artificial myopia. It is to be determined as for natural myopia, and the amount of myopia it represents deducted from the total strength of the lens. The remainder will be the power required to overcome the divergence of the rays, or the strength of lens needed to correct the hyperopia.

For example, suppose the movement of the light in the pupil is found at all distances to be *with* the movement of the light on the face, and on placing a 5 D convex lens before the eye it is found to be still *with* the movement of the light on the face when the examiner approaches to a little within 1 meter, but appears reversed if looked at from a distance slightly greater than 1 meter. The point of reversal then is at 1 meter; 1 D of the strength of the lens is making the rays convergent, while the other 4 D have been used to overcome the divergence of the rays as they came from the eye. Therefore the eye must be 4 D hyperopic. For accuracy it is better here, as in the case of natural myopia, to make the final determination with a lens that brings the point of reversal $\frac{1}{2}$ to 1 meter from the eye.

Application in Emmetropia.—The application of skiascopy for emmetropia is precisely the same as for hyperopia; but it is found that the artificial myopia caused by the convex lens equals the full strength of the lens, proving that the rays must have emerged from the eye parallel.

Application in Regular Astigmatism.—The principles involved and the methods to be employed are essentially the same as in myopia or hyperopia; but the refraction has to be determined in the two principal meridians instead of in any meridian indifferently, as it can be where all meridians are alike. To determine the refraction in a certain meridian the light must be made to move back and forth in that particular meridian by rotating the mirror about an axis at right angles to it.

The direction of either of these principal meridians is revealed by the area of light in the pupil assuming the form of a more or less distinct *band of light*, extending across the pupil in the direction of this meridian, when its point of reversal is approached. This band can be clearly distinguished only when the surgeon's eye is much nearer to the point of reversal for one principal meridian than to the point of reversal for the other principal meridian. In such a position this band is, for the higher degrees of astigmatism, very noticeable, and fixes with the greatest accuracy the direction of the principal meridian. When the band-like appearance is most noticeable, it is easy to cause its apparent movement from side to side; but it is more difficult to distinguish the movement in the direction of the length of the band. Still, this latter movement is the one that must be especially watched, and its reversal point determined.

When the astigmatism is very low, the appearance of a band may be very indistinct, or not at all perceptible. But in such cases it will be found that when the surgeon has reached the point of reversal for move-

ment of the light in one direction, there is still distinct movement, either direct or inverted, in the direction at right angles to this; and he will thus know he has tested one meridian of an astigmatism, and must in the same way ascertain the point of reversal for the other at right angles to it. When the surgeon is closer to the eye than the point of reversal for either meridian, the movement will be *with* the light on the face *in all directions*. When he is at the point of reversal for the meridian which has its point the nearer to the eye, there will be *no distinguishable movement in the direction of the band* visible, but still a *movement (with) at right angles to it*. When he is between the two points of reversal, there will, in the direction of the nearer meridian, be an inverted movement of the light (*movement against*), but in the other meridian a direct movement (*movement with*). When the farther point of reversal is reached, the *direct movement* in its meridian *ceases*, while the movement in the other meridian continues inverted (*against*). When the surgeon has drawn back beyond both points of reversal, the movement is reversed *against the light on the face in all directions*.

Having determined the amount of myopia, natural or artificial, in both principal meridians, the strength of the cylinder required to correct the astigmatism will, of course, be the difference between the refraction for the two meridians. It is well to put this cylinder before the eye to see if it does accurately correct the astigmatism, giving the same point of reversal for all meridians; and, for accuracy, the spheric lens which will bring this point of reversal to the distance of $\frac{1}{2}$ to 1 meter should be used with it.

Application in Irregular Astigmatism.—If the pupil is dilated, it will always be found that the refraction of the eye varies in different parts of it, so that points of reversal for different parts of the pupil lie at different distances in front of the eye. Near the point of reversal both direct and reversed movements of the light are visible at the same time in these different parts of the pupil. Usually there is at the center of the pupil a considerable area that has about the same point of reversal, called the *visual zone*. This is the part through which light will come to be focused on the retina when the eye is in use. For practical purposes it is to the refraction of the visual zone that attention should be paid, the refraction in the other parts of the pupil being of little importance.

When the visual zone of the pupil differs materially in refraction from the part of the pupil that surrounds it, the eye is said to present *aberration*. This is called *positive* when the center of the pupil is more hyperopic or less myopic, and *negative* when the opposite is the case. When the aberration is high, on examining it from near the point of reversal of the margin of the pupil, the movement of the light will be swift at the margin and slow in the center, making it look as if the light in the pupil were wheeling around a fixed point at the center. This appearance is marked in conical cornea. Aberration of moderate degree causes the appearance of a ring of light at the margin of the pupil, which has a very distinct movement when the point of reversal for the center of the pupil has been reached.

Measurement of Accommodation by Skiascopy.—The near point of accommodation can be determined by having the patient fix the upper edge of the plane mirror, or the forehead just above it, and approaching his eye until the movement of light and shadow in his pupil is clearly *with* that of the mirror, in spite of his strongest effort to accommodate for the distance of the mirror. The point at which this direct movement cannot be overcome by accommodation is the *near point.*

This can be tested when the eye is free from the influence of a cycloplegic, either before any measurement of the refraction has been made, or after the refraction has been determined and the correcting lenses placed before the eyes. In the latter case the distance of the near point from the eye will be the focal distance of the lens equalling the accommodation. If the light in the pupil begins to move *with* the light on the face at $\frac{1}{3}$ meter, although a strong effort of accommodation is made, as shown by the convergence of the visual axes, the total accommodation is 3 D.

Letters may be placed on the upper part of the mirror plate, or on a special card, to give the patient a definite object to fix upon. Such an object may be placed nearer to the patient or farther away than the observer's eye. The lenses required to neutralize the movement of light and shade in the pupil when the patient's gaze is fixed at a certain distance, as at 1 meter, measure the *relative accommodation* for that amount of convergence. This method of testing the eyes has been elaborated by Cross, Sheard, and others into a method which is called *dynamic skiametry.*

Conditions of Accuracy.—The feebleness of the light in the pupil, when the eye is viewed from near its point of reversal, makes it important to have the source of light as bright as possible. The fact that near the point of reversal the light area on the retina is greatly magnified makes a small source of light desirable. Direct sunlight is ideal, but is not generally available. An excellent source of light is a Shahan lamp with a diaphragm placed in front of it having a 2 mm. opening.[1]

To bring out most distinctly the band of light in the pupil, characteristic of regular astigmatism, it is necessary that the light thrown in the eye should be perfectly focused on the retina in the direction at right angles to the band. This is accomplished when the source of light (reflected in the mirror) is at the point of reversal for the principal meridian where the band seems to move from side to side with the light on the face, and the source of light far enough from the mirror to have its reflection located at the point of reversal for the other principal meridian of astigmatism.

The amount of hyperopia or myopia can be most certainly ascertained when the eye is under a cycloplegic. But the presence of regular astigmatism and its principal meridians is most distinct when the pupil

[1] To get the full benefit of a small source of light the sight hole in the mirror should be small—2 mm. or less in diameter; and the glass should not be cut out of the sight hole, merely having the silvering removed from this space. This modification of skiascopy has been called objective *stigmatoscopy* (Gullstrand).

is not so widely dilated as to bring in the irregular astigmatism and aberration always present in the margin of a dilated pupil. A pupil 5 or 6 mm. in diameter is most favorable.

Cycloplegics and Mydriatics.[1]—In addition to the use of the mydriatics in the treatment of diseases of the eye—*e. g.*, iritis—these drugs are employed as aids in an accurate determination of ametropia. With the ophthalmometer and by obtaining the manifest correction good results may be obtained; but in all patients of suitable age, and in the absence of contraindicating symptoms, an active mydriatic should be employed in the measurement of all errors of refraction. The mydriatic (cycloplegic) accomplishes three purposes:

1. It dilates the pupil, and permits a thorough exploration of the interior of the eye, as well as a more perfect examination of the lens and vitreous humor than could be obtained without its aid. The student should not, of course, think it necessary to dilate the pupil of each eye which he subjects to an ophthalmoscopic examination; but glasses should not be adjusted without a thorough knowledge on the part of the examiner of all the details of the eye-ground and the transparent media.

2. It paralyzes the action of the ciliary muscle and places the accommodation in abeyance, rendering manifest types of ametropia which otherwise would remain latent.

3. It fulfils the important function of giving, if its action is prolonged, as for example, with atropin, physiologic rest to the eye that is under its influence, and consequently helps to subdue any retinochoroidal disturbance or other congestive condition that pre-existing eye-strain may have originated.

In practice, various mydriatic (cycloplegic) drugs are employed, the most common being the sulphates of atropin, hyoscyamin, and duboisin, and the hydrobromid of homatropin and scopolamin.

(*a*) *Atropin.*—Atropin sulphate is usually employed in a strength of 4 grains (0.26 gm.) to the ounce (30 c.c.). A drop of such a solution dilates the pupil in about fifteen minutes, and a very few moments later begins to paralyze the accommodation, which sustains a full paralysis in about two hours. The effect of atropin upon the accommodation remains for a week, but if, as is commonly the case, the drug is used for several days at a time, this influence is much prolonged, and full return to the previous power of accommodation is not secured for about twelve or fourteen days.

In using atropin sulphate for the purpose of correcting errors of refraction, a solution of the strength given above should be instilled into the eye, one drop at a time, three times for at least a day, preparatory to the determination, and in young subjects possessing hyperopic eyes, with active ciliary muscles, especially if there is associated spasm of accommodation, the drug must be continued for several days, or even longer, before the desired result is reached.

[1] The terms "cycloplegic" and "mydriatic" are constantly used synonymously, although some of the mydriatics have little or no influence on the ciliary muscle.

(*b*) *Hyoscyamin* is usually employed in the strength of 2 grains (0.13 gm.) to the ounce (30 c.c.) in the same manner. It produces wide dilation of the pupil and complete ciliary paralysis, the effect of which lasts from six to seven days. Many surgeons prefer this drug to atropin, and believe that its effects are equally good, while it enjoys the advantage of a much more temporary action upon the function of the ciliary muscle. The salt must be neutral, and the solution filtered through neutral paper (Risley).

(*c*) *Hyoscin* and *duboisin* in similar strength have similar actions, the latter drug being even more transitory than hyoscyamin in its effect, return to accommodative power occurring in from four to five days. Both of them have the disadvantage of producing marked constitutional disturbances, at times rendering their employment disadvantageous. Hyoscin is chemically and physiologically identical with scopolamin.

(*d*) *Homatropin* is a drug which produces a transitory effect upon the ciliary muscle, full return of accommodation usually occurring in about fifty hours after the last instillation.

To use this drug properly it must be employed by cumulative instillations in the strength of 8 to 16 grains (0.52–1.04 gm.) to the ounce (30 c.c.), one drop of such solution being used every fifteen minutes for an hour and a half preceding the determination, and then waiting forty minutes. At the end of this time the maximum effect of the drug upon the accommodation is secured. Complete return of accommodation takes place in about seventy-two hours. In the opinion of some surgeons this drug is an insufficient paralyzer of accommodation, but if caution in regard to the cumulative instillations is observed, and the rule given above carefully followed, entirely satisfactory results may be obtained. Its influence may be neutralized by eserin. Some surgeons prefer homatropin in gelatin disk form, associated with cocain, in the determination of errors of refraction. The author has never been able to convince himself of the superiority of these disks over a solution of the drug, and regards the addition of cocain to the solution as a distinct disadvantage.

(*e*) *Scopolamin*, introduced by Raehlmann, may be employed in the strength of 2 grains (0.13 gm.) to the ounce (30 c.c.). Two instillations of 1 drop each forty-five minutes apart are sufficient. Mydriasis begins in twelve, and is complete in thirty minutes; cycloplegia occurs in about forty-five minutes. Full return of accommodation may be expected in from five to six days. Toxic symptoms—staggering, vertigo, drowsiness, and dryness of the throat—may develop in susceptible subjects and the author has abandoned its use as a cycloplegic.

It is not safe to use strong mydriatics in elderly persons, and they must never be employed if there is any symptom of glaucoma. They are usually unnecessary when that age has been reached after which the accommodation is so weakened that hyperopia ceases to be latent, and they are rarely employed after the forty-fifth year; but Hess and Duane have demonstrated that hyperopia may be as latent at the age of forty-

five or fifty as it is in young persons, and, therefore, the need of a mydriatic may be equally important.

Euphthalmin is an active mydriatic in a 5 to 10 per cent. solution. It produces maximum dilatation of the pupil in about fifteen or twenty minutes, and the pupil returns to its normal size in five to six hours. Its influence on accommodation is so slight that it has no practical value as a cycloplegic. It is an admirable agent for producing brief dilatation of the pupil, and, fortunately, it has no perceptible effect upon the cornea. It may be combined with cocain. During the war it was practically impossible to obtain euphthalmin and *eucatropin* was brought forward as a substitute. It is efficient and should be used exactly as euphthalmin (which now is again available) is employed.

Hydrochlorid of cocain, in addition to its anesthetic action, is, in 2 to 4 per cent. solution, an excellent mydriatic, but its effect upon the accommodation is so slight that it is valueless for the purpose of preparing an eye for the estimation of any error of refraction. Repeated instillations of a cocain solution may produce edema of the corneal epithelium and even opacities, which are transient.

Other mydriatic drugs which may be mentioned are *ephedrin homatropin*, 1 : 10 (Groenouw); *mydrol*, 10 per cent.; *methylatropin*, 5 per cent., and *atroscin*. The last-named drug is similar to scopolamin in its action.

CHAPTER IV

NORMAL AND ABNORMAL REFRACTION

The cornea, aqueous humor, crystalline lens, and vitreous body are the media by which rays of light passing into the eye are refracted and brought to a focus with the production of an image on the retina. Because the two surfaces of the cornea are practically parallel and the index of refraction of the cornea and the aqueous humor is the same, the *dioptric apparatus* may be reduced to the anterior surface of the cornea and the anterior and posterior surfaces of the crystalline lens. The cornea is the principal lens when the eye is at rest, and it has a higher refractive power than the crystalline lens; but during maximum accommodative effort the refractive power of the crystalline lens approaches that of the cornea.

Emmetropia.—To the normal eye, which produces a distinct image of distant objects on the retina without accommodative effort, the term *emmetropic* is applied, and the condition may be defined as follows:

Emmetropia is that refractive condition of the eye in which the dioptric media and surfaces of the eye are capable of exact focusing of pencils of rays falling upon them, and the visual axis corresponds exactly with the focal length of the dioptric apparatus when at rest; the far point lies at infinity, and the eye, in its condition of minimum refraction, is adapted to focus parallel rays on the retina. The principal focus lies on the retina.

The emmetropic eye has an average length of about 22 mm., although emmetropia is still possible with a longer or shorter axis if the curvature of the ocular lenses varies in proportion. Emmetropia, although it exists but rarely, is the ideal state of refraction. Such an eye has a range of vision from infinity to its near point (see table, page 39). Glasses are not required for distant vision, neither are they needed for reading or close work until that age is reached when the accommodative power begins to decline inconveniently—*i. e.*, about the forty-fifth year. No great departure from emmetropia can long exist without producing more or less disturbance of the function of vision and of the nutrition of the ocular tissues, or without originating some of the numerous general and reflex symptoms which arise from "eye-strain." To restore the eye, the refraction of which is abnormal, to a condition of emmetropia, or at least one approaching it, constitutes a most important part of the practice of ophthalmology. This will be more readily conceded when it is remembered that emmetropia is comparatively uncommon, occurring in not more than 1.5 to 2 per cent. of properly examined eyes. Some statistics, those of Tenner, for example (quoted by Duane), record 4 per cent. of emmetropia among 4800 school children.

Ametropia *is any departure from emmetropia—that is, the dioptric media and surfaces do not bring to a point the pencils of rays passing through them, or there is not an exact correspondence between the visual axis and the focal length of the dioptric apparatus when at rest. There is no exact focus, or the principal focus does not lie on the retina.*

Ametropia is denominated *axial* when the length of the eyeball is increased or diminished, and *curvature* when, the axis remaining unchanged, the curvature of the surfaces of the eye undergoes variations. Ametropia presents itself under three conditions: (1) *Hypermetropia, hyperopia, far-sightedness,* or *oversightedness;* (2) *myopia, short-sightedness,* or *near-sightedness;* (3) *astigmatism* or *astigmia.*

It is convenient to distinguish the first two classes of ametropia by the relative position of the principal focus to the retina.

Hyperopia *is that form of ametropia in which there is a principal focus to the eye, but the retina is situated in front of it—the visual axis of the eye is shorter than the focal length of the dioptric apparatus when at rest.*

The far point of the eye is negative, and is represented by the point behind the eye to which rays must converge before entering the eye, in order to be united on the retina. The refractive apparatus of the hyperopic eye, in a condition of minimum refraction, is adapted to bring rays converging to this point to a focus on its retina. Rays passing out of a hyperopic eye have a divergence as if they came from this point.

Causes and Varieties.—The eyeball may be abnormally short, constituting *axial hyperopia;* a deficiency of 1 mm. in the length of the optic axis produces 3 diopters of hyperopia; or its refractive power may be deficient, *curvature hyperopia;* an increase of 1 mm. in the length of the radius of curvature of the cornea produces a hyperopia of 6 diopters; or the crystalline lens may be absent, *aphakial hyperopia.*

Hyperopia is further divided into: (1) *Manifest;* (2) *latent;* (3) *total.* Manifest hyperopia (H. m.) is represented by the strongest convex lens through which an eye with perfectly intact accommodative power retains distinct distant vision; latent hyperopia (H. l.) is the amount in excess of the manifest which can be developed by the use of a cycloplegic—for example, atropin; total hyperopia (H. t.) is the sum of the manifest and the latent—that is, the entire amount of the hyperopia which is developed after paralysis of accommodation or complete relaxation of the ciliary muscle. Evidently latent hyperopia is the difference between the manifest and the total. Manifest hyperopia is either *facultative* or *absolute*—facultative when it can be overcome by an effort of accommodation, absolute when it cannot be overcome by an effort of accommodation.

Hyperopia is nearly always *congenital,* and is often *hereditary,* especially its high grades. In a certain sense it may be regarded as due to an imperfect development of the eyeball, which, however, may increase its length with the growth of the rest of the body, and this refractive condition may diminish, pass into emmetropia, or its approximation, or into myopia. An apparent increase of hyperopia due to failure of accommodation caused by advancing years is often seen—

that is, latent hyperopia becomes manifest. A real tendency to slow increase of hyperopia is due to gradual increase of the size of the crystalline lens. In early life none of the existing hyperopia is absolute unless it is of high degree; after sixty-five practically all of it becomes absolute. Hyperopia due to diminution of the refractive index of the lens in old age is often called *senile hyperopia.*

Symptoms.—Hyperopia renders it difficult to maintain a distinct image of small objects—*e. g.*, printed matter—for prolonged periods of time. If the effort is persisted in, the accommodation becomes exhausted, aching of the eyes and head—in short, the result of *eye-strain*—develop, and finally work must be discontinued (*accommodative asthenopia*). Sudden failure of accommodation, with consequent blurring of vision, is frequent, and often first appears if the patient has been weakened by illness. The subjects of hyperopia not uncommonly place a book or small objects in a strong light in order to contract their pupils and thus render vision clearer.

Hyperopia frequently gives rise to *spasm of the accommodation,* owing to the persistent contraction of the ciliary muscle necessary to overcome this error of refraction, and then simulates myopia, distant vision becoming indistinct. In these circumstances concave lenses may improve vision, and, in ignornace of the true state of affairs, are sometimes prescribed, much to the detriment of the patient. A cycloplegic will reveal the real condition of the refraction. It occurs also in myopic eyes, which appear to be more myopic than they really are. Spasm is prone to occur in individuals of neurasthenic condition, and is a frequent symptom of hysteria, often associated with cramp of convergence; it bears no relation to the vigor of the accommodation, inasmuch as persons with relatively feeble accommodation may have a marked cramp of the ciliary muscle. Therefore spasm of accommodation is not excess of accommodation (see page 41). Spasm of accommodation is not confined to young subjects, but may occur, and in stubborn form, after the fortieth year of life. It may be produced in normal eyes by the instillation of a solution of an active miotic.

Insufficient power of accommodation, *i. e., subnormal accommodation,* may give rise to marked asthenopia. It is attributed by Theobald to a congenital insufficiency of the ciliary muscle (see also page 41), and its determination and correction in refractive work is most important.

Convergent strabismus is often associated with hyperopia in childhood; it arises in connection with efforts of accommodation (see page 626). When the hyperopia is too great to be managed by the accommodation, the affected children frequently hold their books close to their eyes, and, by contracting the palpebral fissures, are enabled to see better than with the book at a greater distance, because the object is seen under a larger visual angle, and the narrow slit between the lids cuts off the more divergent rays. These children are often erroneously supposed to be near-sighted, and concave glasses are given to them, which increase, instead of mitigating, the trouble.

As a result of hyperopia the coats of the eye become inflamed. Conjunctivitis, blepharitis, styes, chalazia, and congestion of the retina and choroid are very frequent complications. *Persistent headache*, aggravated by using the eyes, various nervous symptoms, reflex in their nature, as well as disturbances in the visual function, are the common results of hyperopia (see also page 154).

Determination of Hyperopia.—Hyperopia always exists: If distant vision is not made worse by a convex glass; if the patient can read fine print through a convex glass at a greater distance than its focal length; if with the ophthalmoscope the interior of the eye, otherwise normal, is seen distinctly with a convex lens; usually if the near point lies at a greater distance from the eye than is proper for the age; and if the phenomena described in connection with the shadow-test on page 123 are present. To ascertain the presence of latent hyperopia a cycloplegic should be employed. Its use is imperative in the presence of spasm of accommodation. The ciliary muscle is fully developed in hyperopic eyes, especially the circular fibers, which may be overdeveloped.

Correction of Hyperopia.—The principal focus, F, of the hyperopic eye lies behind the retina. Consequently the retina, R, is situated

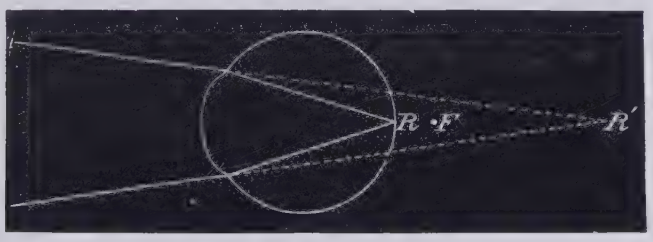


FIG. 62.—Far point of a hyperopic eye. Rays from R on the retina of the hyperopic eye after refraction diverge; these rays, prolonged backward, would unite at the point R'. R' is the far point.

within the principal focus, and its conjugate focus or far point, R', is virtual (Fig. 62). Rays from R seem, after refraction by the eye, to have come from R'; conversely, rays converging to R', after refraction by the eye, unite in R on the retina. The rays which come from the retina, R, of such an eye, after emerging from the eye are divergent, and, prolonged backward, would unite in the point R'. The distance of this point from the cornea is the focal length of the glass which corrects the hyperopia. The amount of divergence of the emergent rays is dependent on the degree of the hyperopia—that is, the distance R lies in front of F. The higher the degree of hyperopia is, the farther R lies in front of F, and the nearer the point of divergence R' lies to R; conversely, the lower the degree of hyperopia is, the nearer the point R lies to F, and the farther back the point R' lies. The distance of R' must be less than infinity; otherwise, the eye would be emmetropic.

If parallel rays are given a convergence to the point R' by a convex lens placed before the eye, the rays will come to a focus at the point R on the retina, since the path of the rays passing into the eye after refrac-

tion by a convex lens (Fig. 63) is exactly the same as that of the rays diverging from the retina and passing outward (see Fig. 62), only the direction is reversed. The far point, R', of the hyperopic eye is the point to which parallel rays must be given a convergence by a convex lens in order to come to a focus on the retina. The amount of this necessary convergence represents the deficiency between the refraction of the hyperopic and that of the emmetropic eye; the degree of hyperopia is, therefore, in an inverse ratio to the distance of R'.

To correct hyperopia the refraction of the eye must be increased by a convex lens of sufficient strength to bring F on the retina. This glass corrects the hyperopia by shortening the focal length of the dioptric apparatus to correspond exactly with the length of the visual axis. The far point R' is removed to infinity. Parallel rays come to a focus on the retina without any effort of accommodation, and rays emerging from the eye are rendered parallel.

In order to neutralize the hyperopia that convex glass must be selected which gives the greatest visual acuteness. As this is obtained when the retinal image is sharply formed, and as this occurs when rays

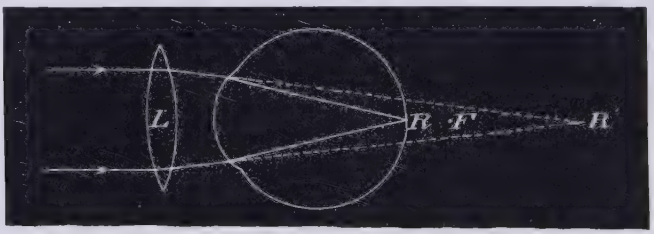


FIG. 63.—Correction of hyperopia by a convex glass. The lens, L, gives to parallel rays a convergence toward the point R'; they will consequently be united on the retina, R. R' is the virtual conjugate focus of R.

are brought to an exact focus on the light-sensitive layer of the retina, the maximum sharpness of sight is the most satisfactory evidence that rays are exactly focused on the retina. If these rays are parallel, the glass which brings them to a focus on the retina corrects the hyperopia. Rays from objects at 6 meters' distance are sufficiently parallel for this purpose.

Correction of Hyperopia with Test-types and Trial-lenses.—The card of test-letters, in good illumination—either artificial light or ample daylight—is hung on a wall, at 4 to 6 meters from the patient. A pair of trial-frames is placed before the patient's eyes, which should be under the influence of a cycloplegic, provided he is not beyond the age when this is necessary, and one eye at a time examined, the other being screened by an opaque disk. He is required to read the smallest letters which he can see distinctly on the card. The resulting sharpness of vision is noted. A convex glass is next placed before the eye. If this glass improves vision, but does not raise it to that which is normal, stronger lenses are tried until the one is obtained which yields the maximum visual acuteness; or, if the stronger glasses do not improve the

vision, successively weaker ones are tried until that glass is found which gives the greatest sharpness of sight. This is the lens which corrects the hyperopia. Even if the acuteness of vision is raised to that which is normal by a convex spheric lens, astigmatism of low degree is not certainly excluded and every eye should be examined with a view to discover any astigmatism. If none exists, the convex glass is all that is required to correct the ametropia.

In the absence of a mydriatic and the presence of some accommodative spasm, vision being equal in the two eyes, a more sutiable glass may often be obtained by testing both eyes simultaneously, because with parallel axes the accommodation is more likely to undergo relaxation. This effect may be further increased by placing a prism of 2° or 3° (centrads) before one eye, with its base inward. The effect of this is to relax the internal recti muscles, and indirectly the accommodation. It is a good plan to begin by placing before the eyes a lens of stronger refraction than the one required, and gradually weakening it by concave glasses of successively higher numbers until normal vision is reached. The glass required is the difference between the two.

The proof that the glass selected is the correct one depends upon the ability of the patient to focus parallel rays on the retina. Parallel rays may be obtained by placing an object at the principal focal distance of a convex lens. The principal focal distance of a 4 D lens is 25 cm. Therefore if the glass corrects the hyperopia, the patient should be able to read fine print at 25 cm. distance with + 4 D added to his correction. If he reads at a greater distance than 25 cm., some hyperopia is still uncorrected. If he reads at a shorter distance than 25 cm., the hyperopia is probably overcorrected.

The degree of hyperopia may also be determined by placing a convex lens before an eye the accommodation of which is paralyzed, and by finding the distance at which small type appears most distinct. Suppose the lens selected is 4 D (focal distance = 25 cm.), and that the patient reads best at 33 cm. Now 33 cm. is farther than the principal focus, and the rays, therefore, are convergent after passing through the lens, since a 3 D lens would render them parallel; 4 D = 3 + 1 would give them a convergence of 1 D to the conjugate focus, 1 meter back of the eye; 1 D, therefore, represents the amount of the hyperopia (see page 136).

Rule.—Subtract from the lens employed the lens whose focal distance equals the distance at which the patient reads. The difference is the degree of hyperopia.

Correction of Hyperopia with the Ophthalmoscope and Shadow-test.—To correct hyperopia in children before they are old enough to read, the ophthalmoscope and skiascopy are the means upon which reliance is placed, but as skiascopy (page 120) is the essentially accurate method of determining the refraction of the eye it should always be employed in childhood as well as in adult life.

Ordering of Glasses.—After the degree of the hyperopia has been determined, the very important question presents itself, What glass

shall be ordered? While the eye is under the influence of the cycloplegic, distant vision is distinct with the full correction; after the effects of the drug have disappeared, it is often dim with the full correction, and a haze seems to lie over all distant objects, which disappears when the glasses are removed. On the other hand, the headache, asthenopia, and congestive troubles return if the hyperopia remains uncorrected. Spasm of accommodation is the disturbing factor in this problem, and it is so variable in different individuals that no precise rule can be given. Many persons wear a full correction with comfort, and do not need any modification; others will tolerate only a small part of the full correcting glass.

There are two methods of dealing with this difficulty: first, to order full correction while the eye is still under the influence of the mydriatic, and to insist that this shall be worn constantly during the time that the accommodation is returning to its normal state. If distant vision remains dim, after full accommodative power has returned, the glasses may be weakened sufficiently to secure normal sight for long ranges.

It should be borne in mind that the glass which gives the best correction at 4 to 6 meters is not the correcting glass for the total H, but, in reality, is an overcorrection of $\frac{1}{4}$ to $\frac{1}{6}$ D. Strictly speaking, rays coming from these distances are not parallel, and the glass which focuses them perfectly on the retina will not perfectly focus parallel rays. Hence, in ordering a full correction, the glass which gives the best vision at 4 or 6 meters must be weakened by $\frac{1}{4}$ to $\frac{1}{6}$ D. If this fact were more often remembered, less difficulty would be experienced in inducing patients to wear a full correction.

Second, the eyes are first allowed to regain their full power of accommodation before the final glass is prescribed, and this is the plan which should be pursued. If vision is normal with the full strength of the glass, it may be ordered; if not, it should be reduced to that number with which full visual acuteness is obtained. This may be only one-half, one-fourth, or even less, of the full amount. It is necessary in these cases to increase the strength of the glass from time to time as symptoms of fatigue manifest themselves. When the glass ordered for distance is only a small part of the full correction, another pair of lenses for reading may be ordered which embodies nearly or quite the full amount of correction.

A frequent cause of inability to wear a full correction depends upon the development of convergence insufficiency, causing an associated action of accommodation with the muscular effort necessary to bring the visual axes into a parallel condition (see page 640).

Other methods are as follows: Instead of ordering the glass nearest in strength to the full correction, with which the patient still has normal vision, the lens which neutralizes the total hyperopia may be reduced by a given amount, usually 0.75 D. Donders advised a glass based upon the manifest H, to which one-quarter of the latent H was added. Macnamara recommends, in absolute hyperopia, the use of a convex glass, the strength of which shall be equal to one-half of the sum of the

manifest and total hyperopia—*e. g.*, manifest H = 1.5 D; total H = 3.5 D; H. m. + H. t. = 5 D; ordered + 2.5 D. Evidently, however, it is not wise to follow a fixed rule.

The amount of the total hyperopia which should be corrected depends upon many factors. The author, if convergence is ample, usually orders the full correction of H less 0.25 D. If there is exophoria, this plan must be modified, or the defect remedied by prisms or by prismatic exercises. The indistinct vision, caused by full correction of H, due to a disturbance of the relative range of accommodation and convergence, may be overcome by systematically training the convergence (see page 643). Whether a glass shall be worn constantly or not depends upon the symptoms which the hyperopia has produced and the character of the patient's work. Frequently hyperopes are entirely comfortable if reading-glasses alone are used. Finally, glasses need not be ordered simply because hyperopia exists, especially in children; but only when it gives rise to the symptoms which have been described. Thus, one person may easily manage one or more diopters of H without glasses; another may have all manner of asthenopic and reflex nervous symptoms produced by 1 D of H or even less.

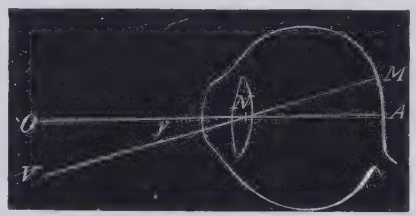


FIG. 64.—Angle gamma in hyperopia: *O–A*, The optic axis; *N*, the nodal point of lens; *V–M*, the visual line, cuts the cornea at inner side of optic axis; *O–N–V*, the angle gamma, in this case is positive; *M*, the macula.

Occasionally very high degrees of hyperopia are encountered—12–14 D, even as high as 20 D (J. A. Wilson)—and sometimes the reaction to these high grades of H is not conspicuous. During the war a number of examples of this character came under the author's observation while revising the ocular examinations of recruits, and in some instances several members of the same family were thus affected. The vision of these high hyperopes in no case reached the normal standard even with the best correcting glass.

The visual line is often very much displaced to the inner side of the cornea in hyperopia, causing a very large value of the angle gamma.

Myopia *is that form of ametropia in which pencils of rays are perfectly focused, but the retina is situated behind the principal focus of the eye, and only those rays which diverge from some point nearer than infinity can come to a focus on the retina. This point is the far point of the myopic eye.*

The far point, therefore, is limited by the amount of divergence necessary to bring the focus of the rays on the retina. The higher the degree of myopia is, the closer will the far point *r* lie to the eye. Rays coming from the retina converge to the far point and form there an image (Fig. 65). This image can be seen by the ophthalmoscope. The far point and the retina are conjugate foci (see page 122).

Cause and Varieties.—Myopia may be produced by increased refraction of the cornea or crystalline lens, *curvature myopia*, or by too great a length of the optic axis, *axial myopia*. In the majority of cases

myopia is due to elongation of the optic axis, often the result of pathologic changes in the coats of the eye.

Myopia may also be occasioned by changes in the shape of the cornea as a result of disease—for example, conical cornea in which case there is also high astigmatism, or, as a transitory condition (see also page 338) in iritis and iridocyclitis. *Corneal opacities* are a frequent cause of myopia. According to Frenkel, bilateral opacities usually produce bilateral myopia, while unilateral opacities more frequently give rise to unilateral myopia, which may affect either eye, according as the one or other eye is most used for near vision. The myopia thus caused appears to be an axial and not a curvature myopia. Myopia, unlike hyperopia, is rarely congenital. It usually makes its appearance from the eighth to the tenth year, especially during the early school years, hence the term *school myopia*, and it tends to be progressive. Sometimes it is the continuation of a process started in hyperopic eyes, especially in those with astigmatism, and the gradual transition from hyperopia to myopia is not infrequently seen among patients who return

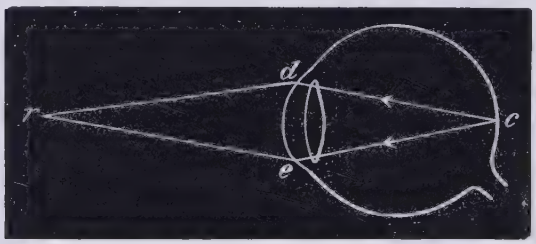


FIG. 65.—Far point of a myopic eye. Rays diverging from the retina, *c*, will, after refraction, converge to *r*; conversely, rays diverging from *r* will, after refraction, converge to *c*; *r* is the far point; *r* and *c* are also conjugate foci.

for examination. The increase of myopia may be arrested as the result of treatment and the optical correction of ametropia.

Myopia is more prevalent in some countries than in others, and is especially frequent in Germany, in the higher classes of the schools, reaching, according to Cohn, 60 per cent. Myopia is said to be more common among Jews than among Christians of the same social class (Sydney Stephenson). Sattler, however, is unconvinced that there really exists a racial inclination to myopia. Harman's statistics indicate that most of the cases of myopia in early childhood occur in boys, but the highest degrees appear in girls. Although the largest number of myopes is found among the upper classes—that is, among those upon whom the demands of modern civilization fall most heavily, and among artisans whose work requires close inspection—high grades of this refractive defect may also be found among those who apparently do not use their eyes for close work, and occasionally among illiterates and among children who have not yet been subjected to the influence of school life. It may be that these subjects of myopia have been

reared under conditions in which they have been obliged to devote themselves assiduously to work at very near range. This seems to be the case among those Chinese of the poorer classes who labor in most unfavorable surroundings, illumination and hygienic conditions being very unsatisfactory. It has been suggested that the high degrees of myopia not uncommonly encountered among Bedouins, Egyptians, and Chinese may be due to the frequency of corneal opacities among them.

Myopia is frequently hereditary, and may occur in several members of one family. Harman found hereditary transmission in 9 per cent. of the cases he investigated. It has been stated that the transmission is more frequent to daughters than to sons. With a strong predisposition to myopia the elongation of the eyeball may take place under comparatively unimportant exertion. Although myopia usually begins in childhood, there may be a *late development* of this refractive error, that is, it may occur after the twentieth year of life. This late development of myopia has been observed in association with constitutional disturbances, with malaria (T. Becker), with goiter and obesity (R. Wirtz), and with pituitary body struma.

That acute posterior scleroticochoroiditis may occasion myopia, especially posterior staphyloma, as originally taught by von Graefe, is not admitted by Sattler, that is, that it is the cause of the myopia. Obvious choroiditic changes, according to him, are to be considered as a complication which etiologically has nothing to do with the progression of the myopia, although he admits that choroiditis may give rise to a rapid increase and pernicious course in myopia. The author has published some observations which indicate that, as the result of severe choroiditis, myopia may rapidly develop in certain cases, observations which are in accord with the statements of Knies, Priestley Smith, and others. Occasionally after an acute illness, especially one of the exanthemata, myopia develops, or there may be a sudden increase in a pre-existing myopia. Increase in the density of the lens as the result of beginning cataract may cause myopia (*index myopia*), the so-called *second sight* (see page 446), and, according to Hirschberg, the late development of myopia—that is, after the fortieth year, unassociated with cataract formation—is not an uncommon sign of diabetes. *Transitory increase of hyperopia* has also been observed in diabetes due to changes in the lens (Lundsgaard).

In normal eyes the sclera does not yield to the intra-ocular pressure, but if from any cause its resisting power is reduced, distention takes place and the anteroposterior axis of the eyeball is elongated. Among the causes which have been invoked to explain the elongation of this axis of the eye—*i. e.*, the production of myopia—are the following: The incentive given by the shape and size of the orbit to greater development of the eyeball; the compression of the eyeball by the exterior muscles, causing distention of its coats backward on account of the excessive convergence rendered necessary by the close range at which

myopes are obliged to work;[1] the strain of accommodation; racial peculiarities; inflammatory changes within the eye—for example, scleroticochoroiditis, induced by habits of life which promote fulness of the veins of the head and neck and hinder the egress of the blood from the eye or are set up by eye-strain itself induced by excessive study, bad ocular hygiene, imperfect illumination, etc.; and an inherited tendency and a nutritive defect in the sclera, the commencement and increase of the myopia being caused by general and local vascular congestion, which are the result of constitutional disturbance—for example, cardiovascular and nasal disease (Batten); increased intra-ocular tension and distention of the eyeball posteriorly due to obstruction to the outflow of lymph from the lymph spaces between the retina and choroid into the lymph spaces of the optic nerve (Edridge Green); retardation of the outflow from the eye due to congenital deficiency of the ciliary muscle which causes increased intra-ocular pressure and expansion of the eye which is distensible during early life (A. Wood).

Prolonged use of the eyes at near work necessitating excessive convergence and muscle pressure has been invoked to explain the acquisition of myopia in many cases, but only a portion of those subjected to such a strain become myopic. Indeed, there is sufficient clinical evidence to show that overuse of the eyes and excessive accommodation or convergence are not the main factors in the development of myopia. Therefore, as Fuchs remarks, special additional factors must be present: predisposition, improper ocular and general hygiene, spasm of accommodation, and, especially, astigmatism. Irregular, inverse, and oblique astigmatism are of marked significance in this respect. In the study of myopia a disturbed balance of nutritive factors must be given due consideration.

Among other causes of less moment may be mentioned an unusually great distance between the pupils, rendering convergence more difficult, a divergent squint, and a large size of the angle gamma (in this case negative), demanding more strain on the part of the eye muscles in the efforts of convergence. After myopia is once produced the eyeball, by its oval shape and greater size, may act as a cause of the further development of this refractive defect by reason of the increased muscular effort which is required to rotate such a globe inward during convergence, and the compressing effect of the external recti muscles on the increased posterior segment of the eyeball.

At first probably all cases of myopia are *progressive*, but many are checked because the eyes are removed from the strain of close work or are placed under better hygienic surroundings; that is, the myopia becomes *stationary*. Other cases may progress until the increased effort

[1] Compression of the eyeball in these circumstances may be caused by the external rectus. According to Stilling, the superior oblique is the principal compressing muscle in myopia, the low position of the trochlea increasing the amount of force which this muscle exercises on the globe. Schmidt-Rimpler rejected Stilling's conclusions, and Hamberger's measurements do not confirm Stilling's contention that in myopia the vertical diameter of the orbit is decreased.

of convergence demanded by the increased myopia becomes too difficult to sustain, one eye deviates outward and there is produced a *divergent strabismus*. Then further increase of myopia may stop, or the inflammatory changes already set up within the eye may continue, the distention of the ocular coats increases, and the most serious organic lesions arise; in other words, there is *malignant* or *pernicious myopia*.

Symptoms.—The symptoms of myopia naturally arrange themselves into two classes, subjective and objective.

The *subjective symptoms* are those which arise because the range of vision is limited by a radius of a few centimeters. Distant objects are not clearly perceived by the myopic patient, because as soon as an object passes beyond his far point it becomes indistinct. According to Seggel, the *light-sense* diminishes with an increase of myopia. Percival Hay finds that if refractive errors are low, they do not affect the light-sense, but if they are high they tend to increase the light-difference.

Many myopes have an inclination to avoid outdoor sports on account of their poor vision, and exhibit a greater fondness for occupations which come within their range—*e. g.*, reading, drawing, etc.—than for others which require good distant vision. The prolonged congestion of the eyes which such habits entail tends to increase the myopia. Headache and reflex phenomena are unusual accompaniments of myopia unless complicated with *astigmatism*, which is an important factor in the further increase of the refraction. Myopia, however, frequently causes aching of the eyeballs, very imperfect ocular endurance, congestion of the conjunctiva—indeed, many of the symptoms which are strictly asthenopic, especially when the choroid is undergoing the changes which are determining the increase in the refractive power. That the full enjoyment of outdoor sports is not at all incompatible with the existence of myopia properly corrected is well known, and it is an interesting fact, pointed out by Goldberg, that many excellent marksmen are myopic.

The *objective symptoms* of high myopia may embrace: (1) A notably prominent and elongated eyeball, with a large and somewhat sluggish pupil; (2) a rather stupid expression of the countenance from inability to note the expression in the face of others; (3) a peculiar manner of reading—the book is held stationary and the face is moved from side to side, following each line; (4) certain characteristic ophthalmoscopic appearances. With the direct method the optic disk appears enlarged; at its outer side there often is a crescentic area of whitish hue, depending upon alterations in the choroid, known as a *conus* or *myopic crescent*. This area may begin next to the disk with a space of complete atrophy, succeeded by a rim of partial atrophy and pigment disturbance, which in its turn merges into a patch of choroidal congestion (*posterior staphyloma*). Sometimes the entire disk is surrounded by areas of choroidal disturbance, and the general choroid may exhibit many alterations depending on congestion, edema, rarefaction, atrophy, and pigment accumulation (see also page 142 and Fig. 66). Weiss and B. Alex.

Randall have described a curvilinear reflex, generally at the nasal side of the disk, as a prodromal sign of myopia. (5) Divergent squint. The squinting eye is often amblyopic. Binocular vision does not exist in such a case; the better eye, freed from the necessity of convergence, reads at the far point without any effort, and glasses for reading are sometimes unsatisfactory because the print appears smaller on account of its removal to a distance greater than the far point of the eye.

The visual axis in myopia sometimes passes through the cornea at the outer side of the optic axis; the *angle gamma* is then *negative*, and the eye, in looking at a distant object, turns inward in order to bring the

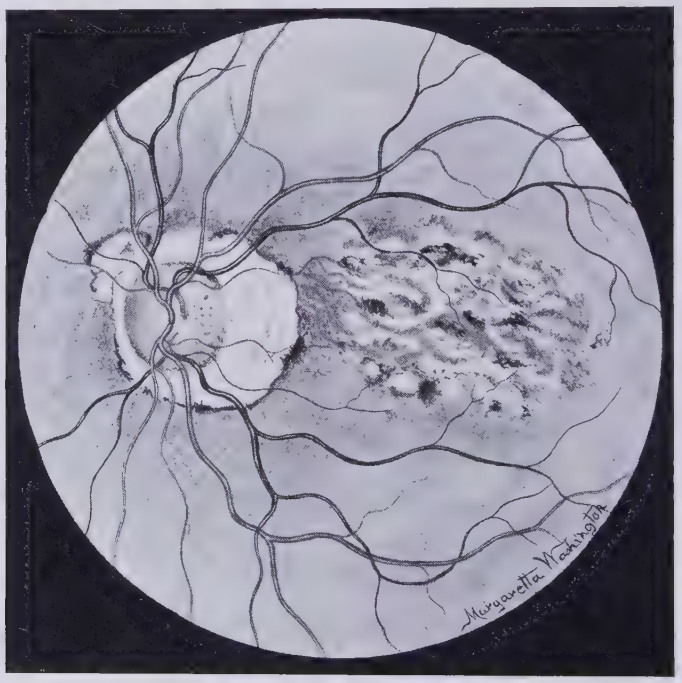


FIG. 66.—Eye-ground in progressive myopia. Large posterior staphyloma surrounding the nerve head. Macular region occupied by areas of semi-atrophic retino-choroidal lesions.

visual line to fix on it, giving rise to an apparent convergent squint (Fig. 67). This renders necessary a greater degree of convergence.

Myopic eyes are popularly considered as strong eyes because they see fine print at close ranges. This is true only in those cases in which the tunics of the eye have suffered no injury—where, for example, the myopia is of moderate degree and the eye-grounds are normal.

Myopia does not usually decrease with age, but, on the contrary, tends to increase up to adult life. With advancing age, however, especially with moderate grades of myopia, a decrease of this refractive defect may be observed.

Very high (10 D and higher) degrees of myopia (*malignant* or *pernicious myopia*) are often marked by ravages in the structure of the choroid and retina. The pigment-cells wander off in some places and accumulate in others, producing marked contrasts in the appearance of the eye-ground. Large areas of atrophy, glistening white in color, alternate with black splotches, and at times hemorrhages occur. The macular region is especially prone to degenerative, atrophic, and hemorrhagic changes. According to Hirschberg, these central changes, characteristic of high myopia, are due to mechanical stretching and not to inflammatory processes. The disk is often surrounded by an atrophic area, the *posterior staphyloma*, which represents an area of thinned and distended sclera.

Posterior staphyloma should not be confused with the conus or myopic crescent, and, according to Schnabel, who accepts von Jaeger's view, it should be regarded as an anomaly in the form of the eye—that is, a malformation and not the result of disease. He believes that there is a connection between retinochoroiditis and posterior staphyloma, but that "the primarily emmetropic or hyperopic eye will not become affected with retinochoroiditis of the macula in consequence

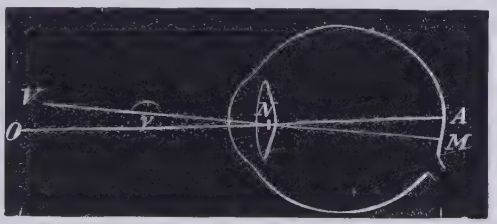


FIG. 67.—Angle gamma in myopia which is negative.

of acquired myopia; only eyes with posterior staphyloma resulting from congenital malformation have, in addition to excessive myopia, an especial predisposition to that grave disorder." The vitreous humor is semifluid, and floating opacities are often visible, sometimes being so large as to obscure vision. Owing to the intimate relation between retinal nutrition and the pigmented epithelium of the retina, the loss of the latter is followed by diminution in the visual acuteness. In high grades of myopia—15 to 20 D, and sometimes still higher—the condition of the eye is desperate, and the morbid processes may culminate in detachment of the retina and complete blindness. Occasionally, in the macular region of myopic eyes may be seen an intensely black area, about the size of the nerve head, with a slightly grayish center, and surrounded by a lighter ring. It stands out well defined. This is the so-called *black spot of the macula in myopia*, first described by Förster and later by Fuchs. It is interpreted in the visual field by a scotoma. The prognosis is unfavorable and the disease may be progressive, although, according to some observers, this lesion renders the eye less liable to the other complications of myopia. Butler and Stargardt have described a central *green spot* in myopia. Occasionally, very high

degrees of myopia, 30 D and more, are encountered, unassociated with the fundus lesions which have been described. Such types of myopia are not progressive and are probably congenital in origin.

Not only is corneal astigmatism a potent factor in the increase of myopia, but, according to Senn, it bears an important relation to central choroiditis and destructive change in the fundus.

In myopia the ciliary body appears to be flat, and the transverse diameter of the ciliary muscle is smaller than it is in the normal eye,



FIG. 68.—Ciliary body of a myopic eye (specimen prepared by Dr. C. M. Hosmer). Notice the abnormally flat appearance of the ciliary body.

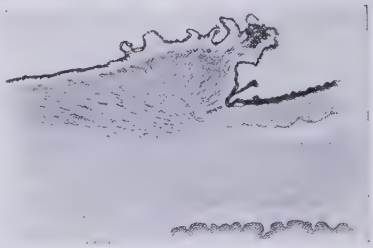


FIG. 69.—Ciliary body of a hyperopic eye (specimen prepared by Dr. C. M. Hosmer).

because its circular fibers, comparatively little employed in the act of accommodation, are poorly developed. On the other hand, certain investigations seem to indicate that the imperfect development of the ciliary body in myopic eyes is congenital and is not the result of the myopia, but may be a determining cause of it. The sinus of the anterior chamber is deeper than it is in emmetropic or hyperopic eyes, and hence the tendency to primary glaucoma in myopic eyes is said to be lessened (compare, however, page 425).

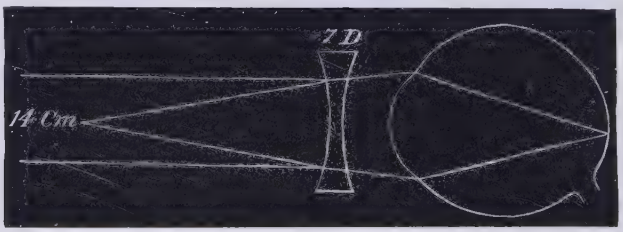


FIG. 70.—Manner in which a concave lens causes rays to diverge from the far point of a myopic eye.

Determination and Correction of Myopia.—Myopia may be determined: (1) By the position of the *punctum proximum* of accommodation, which is closer to the eye than is normal for the age; (2) by the position of the farthest point of distinct vision obtained by test-types; (3) by the ophthalmoscope and retinoscope (page 123); (4) by the concave glass which gives distinct vision at a distance of 4 to 6 meters.

Only those rays which diverge from a distance not greater than the

far point can be focused on the retina of the myopic eye. In order that it shall see at any greater distance than this the rays must be given a divergence as great as if they came from this point (Fig. 70). If the greatest distance at which a myopic eye can see fine print is 14 cm., in order to see at a still greater distance the eye would require a concave glass which would give rays a divergence as if they came from this point. By dividing 100 by 14 we obtain the number of diopters (7) necessary to produce this divergence. As the far point is measured from the cornea, the glass must be placed close to the cornea; if the glass is removed 1 cm. from the cornea, it is plain that its focal point will also be 1 cm. farther away; therefore it is necessary to employ a glass of shorter focus.

Example.—Suppose it is desired to cause the rays to diverge from a point 14 cm. in front of the cornea, and the glass is to be placed at 1.5 cm. in front of the cornea; it is evident, in these circumstances, that the glass would require to have a focus of $14 - 1.50 = 12.5$ cm., or $\frac{100}{12.5} = 8$ diopters.

The proper position for a correcting glass is at the anterior focus of the eye, that is, about 13 mm. in front of the cornea.

In low degrees of myopia this does not affect appreciably the strength of the glass, but in the higher degrees it makes a serious difference. The concave glass is, therefore, somewhat stronger than the actual myopia, especially in the higher grades.

The degree of myopia may be determined approximately by this method more rapidly than by beginning the trial at 6 meters with glasses (in this instance, concave) in the manner already described in connection with hyperopia (see page 134). One example will suffice:

A patient reads fine print distinctly at 8 cm. from the cornea, but not at a greater distance, the eye being under the influence of a cycloplegic; this is its far point. In order that the patient may see at an infinite distance, parallel rays must be given a divergence as if they came from 8 cm. in front of the cornea. If the glass is placed 13 mm. in front of the cornea, its focal length will be 8 cm. — 1.3 cm. = 6.7 cm., or 67 mm.; $\frac{1000}{67}$ mm. equals 15 D as the number of the concave lens required for distant vision. A lens of this number should be placed in the trial-frame, and the vision determined through it by means of test-types at the usual distance. Perhaps a weaker or a stronger lens may give better vision, and hence several numbers should be tried in succession, until that glass is selected with which the greatest acuteness of vision is attained, and which represents the correcting lens.

A patient often will select a glass of higher number than the one really required, because the letters have a blacker and sharper appearance when seen through concave lenses; but unless the stronger glass at the same time secures for the patient an increased acuteness of vision it should be rejected, and the weaker lens adopted. If several lenses give equally good vision, the weakest one should be chosen.

The method of determining the correcting lens in myopia by means

of ophthalmoscopy and skiascopy is elsewhere described (see pages 116 and 120).

Treatment of Myopia.—This should include *prophylactic measures* and the selection of suitable concave glasses. From the eighth to the eighteenth year—that is, during school life—myopia tends to appear and to progress; hence prophylactic means are urgently required during this period. No child should be permitted to begin school duties until the exact state of the refraction has been determined. The systematic examination of children in primary schools, the correction of refractive errors, especially astigmatism and beginning myopia, the elevation of acuteness of vision to the normal standard constitute measures of paramount importance. Strict attention should be paid to the following conditions: A correct position of the head and body during study, secured by means of a suitable desk, the surface of which is so tilted that the page of the book lying on it is parallel to the scholar's face, and by a chair or stool of proper height both in relation to the desk and the floor; the employment of books with sufficiently large and distinctly printed type; good diffuse illumination coming from behind the scholar and preferably over the left shoulder, and therefore the avoidance of glare and sharp contrasts of light and shade; proper ventilation; restriction of the hours of study within reasonable limits and plenty of outdoor exercise. These precautions apply with equal force to hours of study at home.

As Priestley Smith has well said, it is necessary "to suspect every myopia, and especially every youthful myopia, of a tendency to increase, until time has proved it to be stationary; to be doubly suspicious in the presence of congestion or atrophy of the eye-ground; and to reexamine at intervals of six months, twelve months, or longer, according to the nature of the case." These examinations should be made with the help of mydriasis—if possible, with atropin. It is particularly important frequently to investigate the eyes of the children of myopic parents. In some cities—for example, in Berlin—a special curriculum is provided for children with a myopia of 6 D and over (Hirschberg). Harman also urges that children with high degrees of myopia should be instructed in special classes.

A tendency to divergence in early life has sometimes been urged as an indication for tenotomy of the external rectus to prevent the development of myopia. It is questionable whether the procedure is satisfactory in this respect.

Since Fukala's recommendation removal of the crystalline lens (discission, followed by extraction, or *phakolysis*) has been practised by a number of operators for the relief of high myopia (15 D or more). Improvement in vision and increase in the distance at which eyes can be used in near work are the results of successful operations, which, according to von Hippel, may not reach their best standard until a year after the operation—checking of the increase of the myopia.

The chief dangers of the operation are: Intra-ocular hemorrhage, detachment of the retina, secondary glaucoma from swelling of the

lens, iritis, and infection of the corneal wound. The chief contra-indications are: Extensive degeneration of the choroid, retina, or vitreous, diminished intra-ocular tension, a tendency to intra-ocular hemorrhage, previous loss of one eye from any cause, and advanced age. Hirschberg vigorously condemns any tendency to indiscriminate application of the operation and has operated only on carefully selected eyes. (For methods of operating see pages 757, 759). The author's experience with the operation has been very limited, but it has been favorable in those patients whose eyes after careful study have been selected for operation. He believes, however, that in the majority of cases correcting lenses, even in very high grades of myopia, can be made to serve a more useful purpose than operative interference, and has but rarely found it necessary to advise the operation. Hertel has designed *telescopic spectacles*. They somewhat resemble an automobile goggle. They decrease the size of the visual field, but increase the distinctness of distant objects. They are intended to be a substitute for operative procedures in high myopia.[1]

Ordering of Glasses.—After the estimation of the degree of myopia, astigmatism having been excluded, or, if present, corrected, the strength of the glass suitable for constant use, reading, or other special work must be determined. This is decided by the visual acuteness, the accommodation, the degree of the myopia, and the condition of the exterior ocular muscles.

Young people (under twenty) with good vision and a moderate degree of myopia (6 D and under) should wear the full correction constantly if the accommodation is ample and no signs of fatigue are evident.

Indeed, full correction is the object to be attained for young persons with normal visual acuteness and binocular near vision, even with higher grades of myopia, provided the lens selected shall not be an overcorrection when brought close to the eye. A patient wearing a partial correction is tempted to improve distant vision by looking obliquely through the glass. But this gives it a cylindric effect, varying with the direction of the visual axis, and is always injurious. The author is convinced from personal experience that full correction, other things being equal, yields the best results in the management of myopia, and especially in the prevention of its increase, and this conviction is strengthened by the abundant statistical information on this subject which has been collected and analyzed by observers here and abroad. A few authors, for example, Hirschberg, are not in accord with this advice. Hirschberg advises that if the myopia is beyond 3 D, fully correcting glasses should not be worn. Naturally, there are exceptions to the rule, and each case requires thoughtful study; but, in general terms, the full correction of myopia yields the best results because the eyes are placed under conditions which approximate closely the normal both for distant and near vision and the function of accommodation.

[1] See Archiv. f. Ophthal., Bd. lxxv, Heft 3, p. 586, 1910.

Where visual acuteness is imperfect or binocular vision lost, it may be necessary to order a partial correction for near work, and if the patient has attained those years when accommodation naturally fails, he must be provided with lenses for close range, or, if his myopia is of suitable degree, read without glasses.

In high grades of myopia associated with lowered vision it is often necessary to diminish the full correction from 1 to 3 D. It is evident that the greater the visual acuteness, the farther away the same size of type can be seen; hence the demand on accommodation is less as the visual acuteness is greater.

A lack of accommodative power is not infrequently evident when comparatively strong concave glasses are required to correct the existing myopia, but, other things being equal, the discomfort which this may occasion disappears with reasonable rapidity. It may be necessary to give a partial correction for near work until ample power of accommodation is gained, when the full correction should be used for all purposes.

Patients who have long been accustomed to wear partially correcting glasses for all purposes, or to wear a full correction for distance and read without glasses are often quite unwilling to change their habits in these respects. Whether they should be advised to change them naturally depends upon the symptoms which exist, the condition of the eye, and the character of the work required of them. Bifocal glasses —the upper portion being a distance glass and the lower segment a + 1 or 1.5 D—often serve a useful purpose, especially in office work.

As age advances an additional glass should be ordered for reading which will give the patient a far point of from 30 to 60 cm. In order to obtain this the full correction must be diminished from 1.50 to 3 D.

The *position* of the lens used to correct high grades of myopia is of great importance (page 147). The nearer the lens is placed to the cornea, the stronger it becomes; conversely, the farther it is removed from the cornea, the weaker it is. The strong concave lenses necessary to correct high degrees of myopia may sometimes be utilized by the patient to gain artificial accommodation. By bringing them close to the eye vision is adapted for distance; by pushing them from the eye divergence is lessened and the eye is adapted for a closer point.

The visual acuteness in high myopia is usually reduced, and in those cases accompanied by changes in the retina and choroid this reduction assumes a considerable grade. Sometimes very slight improvement in distant vision is secured by concave glasses, and near vision may not be at all benefited. In these circumstances patients see better by using one eye alone and bringing the print or other work close to the eye, because the enlarged retinal image compensates for the diminished visual acuteness. These cases, however, are seldom encountered, and a concave lens, properly selected, almost always improves both near and distant vision. Again, many persons require sharp distant vision and are naturally inconvenienced in its absence.

Therefore it must be remembered that the glass selected while the eye is under the influence of a cycloplegic, should the selection have been made at 4 meters, for example, will be an undercorrection of the myopia of 0.25 D after the effect of the cycloplegic drug has passed away. The glass must be strengthened by this amount in order to gain the maximum acuteness of vision at long ranges.

Concave glasses diminish the size of the retinal image, especially when the glass is removed farther from the eye. The retinal image is larger in myopia than in emmetropia, but if the correcting lens is exactly 13 mm. in front of the cornea, the image is of the same size as in emmetropia.

Concave lenses act as prisms when the visual line passes through any portion except the optical center. The optical centers should always be separated by a space equal to, and never less than, the interpupillary distance, except in those cases of weakness of the internal rectus muscles where it is advisable to increase the distance between the centers. This produces the effect of a prism with its base inward—that is, it lessens the amount of convergence which otherwise would be required. The deviation may be calculated from the focal distance of the lens and the amount of decentering. The distance the optical center is displaced, divided by the focus, equals the tangent of the angle of deviation. Myopes with decided esophoria often read more comfortably without than with glasses.

The painful glare of light sometimes caused by wearing concave glasses may be modified by tinting them. For this purpose the lighter shade of Crookes' glass serves a useful purpose.

The reading glasses for myopes are described under Presbyopia.

Astigmatism (Astigmia).—In the preceding forms of ametropia, H. and M., the cornea has been considered as an ellipsoid of revolution, so that planes passing through it in various directions, vertical, horizontal, and oblique, produce sections having an equal curvature. Equal refraction, consequently, takes place in these different planes. Variations in the curvature of the different meridians produce differences in their refractive power; in some of these meridians the eye must, therefore, be ametropic. Three conditions may arise:

1. The eye may be emmetropic in one meridian and ametropic (either H. or M.) in the others.

2. The eye may be ametropic (H. or M.) in all meridians, but in different degrees.

3. The eye may be ametropic in all meridians, but in some H. and in others M. (H. and M.).

It is convenient to designate the different parts of the eye by imaginary lines similar to those employed in geography.

The *axis* of the eye is a line drawn from the center of the cornea through the center of the ball. Passing through the center of the lens and the center of rotation, it penetrates the sclera between the optic nerve entrance and the macula. The anterior and posterior extremities of this line are the *poles* of the eye.

A great circle extending round the ball perpendicularly to the axis and at an equal distance from the two poles is called the *equator* of the eye; other great circles passing through the poles are called *meridians.*

The lens is described in a similar way by its axis, anterior and posterior poles, and equator.

When the meridians of the cornea have an equal curvature, the rays of light gather in one common focus. Frequently the cornea has meridians of unequal curvature producing greater refraction in some meridians and less in others. The rays passing through the meridian of highest refraction reach their focus soonest, while those passing through the least refracting meridian come to a focus farther back.

Definition.—*Astigmatism (or astigmia) is an ametropia of curvature in which rays from a luminous point—for example, a star—are not turned toward any single point, but form a diffusive area, the shape of which is a line, an oval, or a circle, according to the situation of the retina (regular astigmatism) or an irregular area (irregular astigmatism).*

Seat of Astigmatism.—Usually the cornea is the seat of astigmatism, but astigmatism may also be produced by an oblique position of the lens (*lenticular astigmatism*), or by the visual line passing eccentrically through the cornea.

When the meridians of the cornea progress evenly in their refraction from the lowest to the highest, the astigmatism is termed *regular.* When the curvature in different parts of the same meridian varies or successive meridians differ irregularly in refraction, or the meridians vary irregularly in their curvature as the result of cicatrices from ulcers or distention of the cornea from inflammation, the astigmatism is called *irregular.*

Almost all eyes possess more or less *irregular astigmatism.* Usually it is only slight, and gives no serious inconvenience for ordinary vision, but all points of light, such as stars, distant street-lamps, etc., shoot out rays and twinkle as the result of the irregular astigmatism of the eye. The seat of this irregular astigmatism is in the crystalline lens (so-called *physiologic astigmatism*). In the lenses of young people the union of the sectors is visible by three faint lines—the *lens star;* in the adult secondary rays are also visible. Slight differences in the density of the several sectors are sufficient to produce a distorted image of a luminous point. Should pathologic conditions arise, for example, beginning cataract, lenticular astigmatism may be much increased (page 446).

Principal Meridians.—In regular astigmatism the cornea has one meridian with the shortest radius of curvature producing the highest refraction, and another meridian, at right angles to this, with the longest radius of curvature and the least refraction. These are called the *principal meridians,* and may be situated in any part of the cornea, but there is a disposition of the greatest refracting meridian to lie in or near a vertical direction, and of the least refracting meridian to lie in a horizontal direction.

If the meridian of greatest refraction is vertical or nearly so, the *astigmatism* is described as *direct* (*astigmatism "with the rule"*); if the

meridian of greatest refraction is horizontal or nearly so, the astigmatism is called *inverse* (*astigmatism "against the rule"*); if the direction of the principal meridians approaches 45° and 135°, the astigmatism is often designated *oblique*.

To simplify the phenomena of astigmatism the principal meridians will be considered as running vertically and horizontally with the greatest refraction in the vertical, and the least refraction in the horizontal, meridian.

Form of the Image of a Point Focused by an Astigmatic Eye.—The rays passing into an astigmatic eye, thus considered, are most sharply refracted by the vertical meridian. The bundle of rays, instead of having a round section, forms a horizontal oval, which becomes smaller as the rays travel farther backward, but the vertical diameter of the oval lessens most rapidly until, when the focus of the vertical meridian is reached, the figure becomes a horizontal line, because all the rays are brought to one level and remain diffused only in the horizontal direction.

Farther back the rays, after passing this focus and crossing, diverge again vertically, and the figure becomes once more a horizontal oval, but shorter because the horizontal diffusion is diminished.



FIG. 71.—Retinal images of a point in the different forms of astigmatism: *A*, Compound hyperopic astigmatism; *B*, simple hyperopic astigmatism; *C*, *D*, *E*, mixed astigmatism; *F*, simple myopic astigmatism; *G*, compound myopic astigmatism.

Still farther, the figure assumes the form of a circle; the diffusion of the horizontal rays has become less, and that of the vertical rays more. The figure becomes next a vertical oval, then a vertical line as the focus of the horizontal meridian is reached. Finally, the section is again a vertical oval, the horizontal rays, having passed their focus, cross and begin to diverge (Fig. 71).

It is evident from this that no matter what position the retina may occupy, no distinct image can be formed upon it, but there must always be overlapping of the images of the different points of an object, causing a blur or a wrong impression of its outline.

The focusing of light by an astigmatic eye is further illustrated by a figure and description borrowed from Edward Jackson as follows: In this figure *V*, *V* represents the meridian of greatest curvature and *H*, *H* the meridian of least curvature. Vertically, in the direction of *V*, *V*, all the rays above and below are turned down and up to such an extent that they are brought to the level of the central ray at *F*. But horizontally, in the direction of *H*, *H*, they are not turned to such an extent by the weaker curvature, and do not come into line with the central ray until they reach *G*. The rays are converged in both directions,

but unequally, so that at *F*, being all gathered to the same level, but still spread out laterally, they form a horizontal focal line *F*, *F*. Then the rays begin to spread up and down, although still gathering together from the sides, until at *G* they are collected in the vertical focal line *G*, *G*. Beyond *G* they spread out in all directions. *F*, *F* is called the *anterior focal line*, and *G*, *G* the *posterior focal line*. The distance between the focal lines is the *focal interval of Sturm*.[1]

Symptoms.—From what has been recorded in the preceding paragraphs it follows that the acuteness of vision is diminished by astigmatism. Letters are not distinctly seen, some letters being confused with others—H and N, B and S, F and P, F and R, W and M, K and X, V and Y. The overlapping of the diffusion areas in the retinal image produces, in high degrees of astigmatism, an apparent doubling of the object. The indistinctness of vision compels a closer approximation of the object, with a consequent strain upon the accommodation. Although low degrees of astigmatism may be compatible with fairly

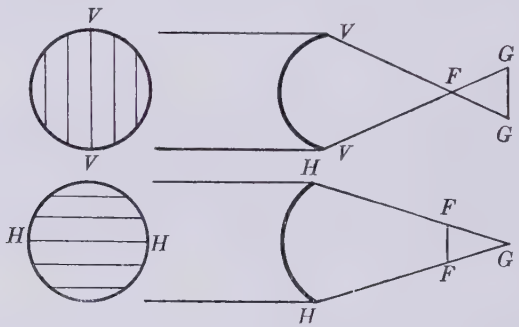


Fig. 72.—Refraction of light in principal meridians of an astigmatic eye, the upper part representing the vertical, and the lower part the horizontal meridian (Jackson).

distinct vision, this does not exclude eye-strain. As the amount of this refractive defect increases the acuteness of vision diminishes. The effort to see clearly and the frequent apparent movement and dancing of small objects, such as letters, add materially to the discomfort of the subjects of astigmatism (see also page 154).

Astigmatic persons learn to overcome their refractive defect by contracting the lids close together in order to make a horizontal slit. The vertically divergent rays are thus excluded, and the eye, accommodated for the horizontally divergent rays, receives a more distinct

[1] Although, as Duane points out, figures "showing Sturm's conoid and Sturm's interval and the diffusion images formed by a spherocylindric surface give only a rough idea of astigmatic refraction," they possess a definite practical value for the student and hence are utilized. Duane refers to Gullstrand's demonstration that the diffusion images formed by a spherocylinder are not the regular lines and ellipses usually pictured, but stellate figures; also "the shape and size of the diffusion image are not of so much consequence in determining the clearness of vision as the way in which the light is concentrated in the diffusion image."

though fainter image. There is an almost characteristic facial expression in astigmatism caused by contraction of the lids.

Astigmatism produces an indistinctness in the appearance of fine lines running in certain directions, the direction of the indistinct lines being determined by that meridian which has its focus on or nearest to the retina. This meridian, therefore, most nearly approaches

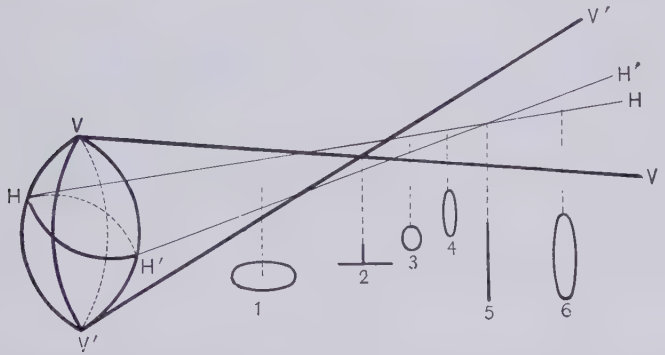


FIG. 73.—Rays passing through an astigmatic lens (Thorington).

emmetropia; the lines parallel to it appear indistinct, while those parallel to the opposite meridian, or the one farthest removed from emmetropia, are most distinctly seen.

In those cases in which the horizontal meridian is emmetropic and the vertical meridian ametropic, fine parallel lines running in a horizontal direction will appear spread out into thick bars, while vertical lines will appear distinct.

To understand this the student should remember that rays diverge from a horizontal line in all directions; those which pass through the horizontal meridian, if they are not exactly focused, spread out in the direction of the line, causing its extremities to appear somewhat faint in outline, but do not blur its width. The rays which diverge in vertical planes from the different points in the line pass through the vertical meridian. If this is not emmetropic, the breadth of the line appears thicker; but if the vertical meridian is emmetropic, it forms a distinct point in the image, of each point in the object, by bringing the rays which pass through it to a focus. A horizontal line thus appears as a succession of distinct points when the vertical meridian is emmetropic. Vertical lines, in the same way, appear most distinct when the horizontal meridian is nearest to emmetropia, or if oblique lines appear most distinct, the meridian at right angles to their direction is the one nearest to emme-

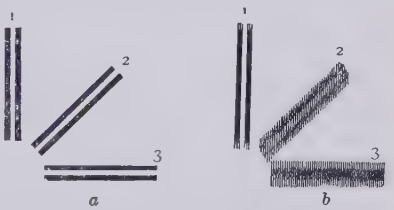


FIG. 74.—Illustrating the appearance of lines running in different directions as seen by (*a*) the normal eye and (*b*) the astigmatic eye (Jackson).

tropia. Luminous points are drawn out in the direction of the ametropic meridian, and luminous circles become elongated into ovals.

Astigmatism may be responsible for the most aggravated types of *asthenopia* and most marked symptoms of *eye-strain*. Fully 70 per cent. of functional *headaches* are caused by this type of refractive error, either alone or in association with other forms of ametropia. The headache may vary from a moderate frontal distress to violent explosions of pain, and may be situated in any portion of the cranium. That true *migraine* is caused by astigmatism alone is doubtful; that the correction of astigmatism is an important, indeed, an essential, part of the treatment of this affection should not be disputed. Furthermore, all manner of reflex nervous disturbances, vertigo, pseudochorea, habit-spasm, epileptiform convulsions, melancholia, neurasthenia, tachycardia, night-terrors, flatulent and other types of dyspepsia, indigestion, and even constipation are the frequent results of astigmatism, not only when the error is of high degree, but commonly, indeed, more commonly when it exists in low grade, and often unassociated with any symptoms which prominently direct attention to the eyes as the cause of the

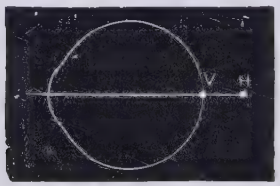


Fig. 75.—Foci of the principal meridians in simple hyperopic astigmatism.

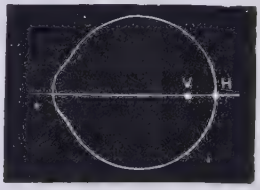


Fig. 76.—Foci of the principal meridians in simple myopic astigmatism.

distress. Pains strangely and persistently situated in the nape of the neck, between and under the shoulder-blades, in the precordium, at the end of the spine, and deep in the mastoid may owe their origin to the same cause. Tilting of the head and shoulders is often the result of astigmatism, and the same refractive anomaly is the exciting cause of some of the cases of lateral curvature of the spine so often seen in young subjects. (See also page 641.)

Regular astigmatism is divided into five varieties, according to the relative position of the retina to the foci of the two principal meridians. The focus of the horizontal meridian is represented by H., that of the vertical meridian by V.

1. **Simple Hyperopic Astigmatism.**—In this variety one meridian, more frequently the vertical, is emmetropic, and the horizontal meridian is hyperopic. The focus of the vertical meridian is on the retina; the focus of the horizontal meridian is behind the retina (Fig. 75); horizontal lines appear distinct.

2. **Simple Myopic Astigmatism.**—The focus of one meridian, frequently the horizontal, is situated on the retina, while the focus of the vertical meridian lies in front of the retina. The vertical meridian is

myopic, and the horizontal meridian emmetropic (Fig. 76); vertical lines appear distinct.

3. **Compound Hyperopic Astigmatism.**—All meridians are hyperopic, but more frequently the horizontal presents the greatest ametropia. The focus of each principal meridian is situated back of the retina, that of the vertical generally being nearest to it (Fig. 77); horizontal lines are usually most distinct.

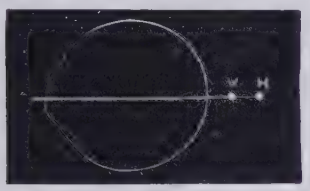


FIG. 77.—Foci of the principal meridians in compound hyperopic astigmatism.

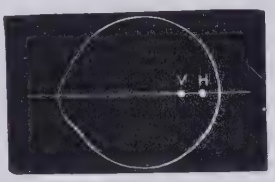


FIG. 78.—Foci of the principal meridians in compound myopic astigmatism.

4. **Compound Myopic Astigmatism.**—All meridians are myopic, but the vertical presents the greatest ametropia. Both principal meridians have their foci in front of the retina, that of the horizontal lying closer to the retina (Fig. 78); vertical lines are usually most distinct.

5. **Mixed Astigmatism.**—The retina lies between the foci of the two principal meridians. The horizontal meridian is hyperopic, and the vertical meridian is myopic (Fig. 79); no lines appear distinct unless the eye simulates myopic astigmatism; in this case the vertical lines appear distinct.

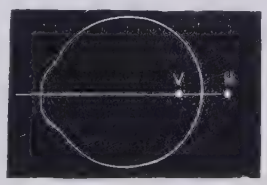


FIG. 79.—Foci of the principal meridians in mixed astigmatism.

It must be remembered, as Jackson has well shown, that astigmatism is likely to change at any period of life and it frequently increases because of the increased asymmetry of the anterior surface of the cornea in childhood. *Inverse astigmatism* is infrequent among children, rarely if ever occurring prior to the tenth year of life; it is probably more common in females than in males. Jackson's statistics, and with these statistics those of the author are in entire accord, indicate that *direct astigmatism* is the exception in the latter part of life when *inverse astigmatism* is much more commonly encountered. John Green, Jr., and W. F. Hardy maintain that inverse astigmatism appears to be nearly as frequent from birth to middle age as from middle age to old age.

Recognition of Astigmatism.—Astigmatism is recognized *subjectively* by the greater distinctness of lines which run in one direction, and the blurring of those lines which run in a direction at right angles to this (see Fig. 74). The vertical strokes of a letter may appear distinct, while the horizontal strokes are hazy.

A diminished visual acuteness unimproved by spheric lenses, in the absence of organic disease of the eye—for example, opacity of the media or lesions of the fundus, or lesions of the visual centers—usually

is due to astigmatism. Letters have a streaked or smeared appearance; a small jet of flame seems to be drawn out in one direction (see also page 152).

Astigmatism is recognized *objectively* by the ophthalmoscope (see pages 116–118), the ophthalmometer (see page 120 and Appendix), and skiascopy (see page 120).

Correction of Astigmatism.—Astigmatism may exist in a very low degree, associated with a much higher degree of hyperopia or myopia, or a marked astigmatism may exist alone, or with ametropia of the other meridians, or finally, mixed astigmatism may be present. There are several methods by which astigmatism may be measured:

1. In all cases of hyperopia or myopia, after the highest visual acuteness has been developed with spheric lenses, and even if the radiating lines on the dial appear equally distinct, a weak convex and a weak concave cylindric lens should be alternately placed in the trial-frame, in addition to the spheric lens, and their axes rotated through 180°.

If, by this maneuver, vision is improved and the patient enabled to read another line of the test-letters, astigmatism is present. For example, if the vision of a case of hyperopia of 3 D is improved by placing in front of the spheric lens a convex 0.50 D cylinder, with its axis vertical, the glass required is + 3 D sph. ⌒ + 0.50 cyl., axis 90° or vertical; but if in the same case the maximum vision previously obtained by + 3 D sph. is not improved by the addition of a convex cylindric lens, a concave cylindric lens should be rotated through 180° in front of the spheric lens. If, in these circumstances, a concave cylinder of 0.50 D with its axis at 180° is found to improve vision and equalize the lines, the formula is + 3 D sph. ⌒ — 0.50 D cyl., axis 180°. This result may be expressed in a simpler form by the formula + 2.50 D sph. ⌒ + 0.50 D cyl., axis 90° (see page 32).

From this it is evident that any spherocylindric combination, in which the spheric is designated by a plus (+) and the cylinder by a minus (—) sign, unless the cylinder is stronger than the spheric, can be reduced to a simpler form, obtained by subtracting the value of the cylinder from that of the spheric lens; the difference is the strength of the required spheric lens. A cylinder of the same strength as the one first employed, with its sign changed to correspond to that of the spheric lens, and the axis reversed, completes the process. This method of correcting astigmatism is best adapted to those cases in which the degree is 0.75 D or less.

2. The position of the principal meridians is determined by means of the clock-face, Snellen's dial, or a series of lines, as is shown in Fig. 80, and especially well with Lancaster's charts.

The most distinct lines correspond to the most ametropic meridian; therefore a *stenopaic slit* is inserted in the trial-frame, in a direction at right angles to this. If vision is normal in this direction, the meridian must be emmetropic and the astigmatism is simple. The slit is then turned at right angles to its previous direction, and the glass found

which gives the highest vision. The astigmatism is represented by this glass. The following are examples:

Simple Hyperopic Astigmatism.—The patient sees horizontal lines most distinctly; the stenopaic slit is placed vertically in front of the eye: and through this $V = \frac{6}{6}$; with the stenopaic slit horizontally placed, $V = \frac{6}{9}$, with + 1 D added, $V = \frac{6}{6}$; hence + 1 D cyl., axis 90°, is the glass required.

Simple Myopic Astigmatism.—The patient sees vertical lines most distinctly; the slit is placed horizontally: $V = \frac{6}{6}$; with the slit placed vertically: $V = \frac{6}{12}$; with — 1.50 added, $V = \frac{6}{6}$; hence — 1.50 cyl., axis 180°, is the glass required.

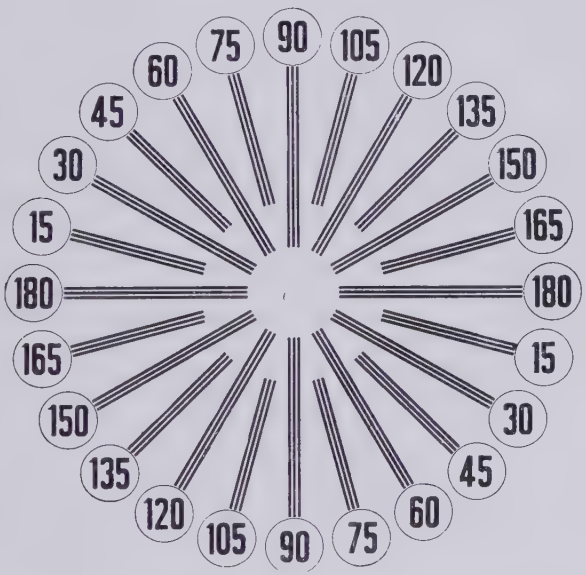


FIG. 80.—Wallace's astigmatic chart reduced to one-sixth of its diameter.

3. The patient may not perceive any difference in the distinctness of the radiating lines until a spheric lens is placed in front of the eye, when some of them become more distinct than the others. The slit is now introduced in a direction at right angles to the distinct lines. Vision is not normal, but a spheric lens improves it, and that lens which gives the best vision with the slit in this direction is selected. The slit is then reversed. The visual acuteness is less through the slit in this position than in the previous one, and a higher lens is necessary to secure the best vision. The astigmatism is represented by the difference between the stronger and the weaker lens. This is an example of compound astigmatism, and is corrected by a spheric lens of the same strength as that which neutralizes the lesser ametropic meridian, and a cylindric lens equal to the difference between the two meridians. The following are examples:

Compound Hyperopic Astigmatism.—No lines appear distinct, or perhaps the horizontal ones only slightly more distinct than the others, but a convex glass makes the horizontal lines decidedly more distinct. The slit is introduced in a vertical direction: $V = \frac{6}{12}$; with + 1.50 spheric lens added, $V = \frac{6}{6}$. The slit is now turned in a horizontal direction: $V = \frac{6}{30}$ with + 3.50 D sph. added, $V = \frac{6}{6}$. The glass required for such a case is + 1.50 D sph. ⊂ + 2 D cyl., axis 90°.

Compound Myopic Astigmatism.—No lines are distinct, but a concave spheric lens possibly makes the vertical lines more distinct than the others, if the visual acuteness is not too much lowered. The slit is introduced in the horizontal direction: $V = \frac{6}{60}$; with — 5 D added, $V = \frac{6}{12}$. The slit is now placed vertically: $V = \frac{6}{60}$, — 7 D is added, and V rises to $\frac{6}{12}$. The glass required is — 5 D sph. ⊂ — 2 D cyl., axis 180°.

Mixed Astigmatism.—Hyperopia exists in one principal meridian, and myopia in the other. Usually no set of lines appears plainer than the rest, but the addition of a concave or convex spheric lens brings out some lines more distinctly than the others. Thus a clue to the principal meridians is obtained. With the slit before the eye, a convex spheric lens is placed in position and the slit rotated until the vision becomes more distinct. The hyperopic meridian has then been found.

Example.—Suppose the hyperopic meridian to be horizontal and V to be most improved by + 3 D. The slit is turned to the vertical position, and it is found that a — 4 D gives the best vision. The difference between these two meridians is 7 D. A + 7 cylinder, axis 90°, placed before such an eye, would produce a myopia of 4 D, while a — 7 cylinder, axis 180°, would produce a hyperopia of 3 D, consequently with the + 7 cylinder we must associate a — 4 spheric lens, and with the — 7 D cylinder a + 3 D spheric lens. Such a case could be corrected by either of the following formulas: + 3 D sph. ⊂ — 7 D cyl., axis 180°; or — 4 D sph. ⊂ + 7 D cyl., axis 90°; or by means of two cylindric lenses with their axes at right angles to each other, viz., + 3 D cyl., axis 90° ⊂ — 4 D cyl., axis 180° (see also page 32).

Dr. J. S. Johnson,[1] of St. Paul, employs a method of determining astigmatism which he calls "the reversal of the clock-dial chart" when it is viewed through successive spheric lenses. The first indication of such a change marks the dividing line between the hyperopia and the astigmatism and between the spheric and cylindric correction. If carried carefully to the point of complete reversal, it will also show the ametropia of highest degree and thus serve all the purposes of the stenopaic slit.

Thorington also utilizes this method in determining the acuteness of vision, which he maintains under definite conditions is an index of the strength of the spheric lens which will give normal vision. For this purpose he has designed a series of "metric test-letters and lines."

The *astigmatic lens*, or *crossed cylinder*, as pointed out by Edward Jackson, is most useful to determine the amount and also the principal

[1] Ophthalmic Record, October, 1901.

meridians of astigmatism. This astigmatic lens is employed as a supplementary lens; the axis of the cylinder is first placed in the same direction as the axis of the cylinder in the trial-frame, and next it is turned perpendicular to it. In one position it enhances the effect of the cylinder in the trial-frame; in the other it diminishes the effect. Therefore, if the vision is unchanged by an astigmatic lens in either of these positions, the cylinder in the trial-frame is correct. If the vision is improved by the astigmatic lens when placed in one position, but not made better in the other position, the cylinder in the trial-frame must be changed accordingly.[1] Various astigmatic lenses may be used having a value of 1 D (+ 50 cyl. ⊂ — 0.50 cyl.), 0.50 D, and 0.25 D.

The following additional facts concerning lenses require mention: If a spherocylinder is in position before an eye, and vision is improved by placing before it another cylinder of the same sign (+ or —), with its axis at right angles to that of the first, it shows that a stronger spheric and weaker cylinder are required.

If vision is improved by placing in position another cylinder of the same sign, with its axis parallel to the first, it shows that the same spheric with a stronger cylinder should be adopted.

If vision is improved by placing in position a cylinder of different sign, with its axis parallel to the first, it shows that a weaker cylinder with the same spheric lens is needed.

If vision is improved by placing in position another cylinder of different sign, with its axis at right angles to the first, it shows that a weaker spheric lens with a stronger cylinder must be employed.

4. Astigmatism is best estimated and the correcting glass determined by *objective methods:* skiascopy and the ophthalmometer. These have been referred to and are elsewhere explained (see pages 120 and 124).

All methods should be tried before the glass is finally ordered, and the highest visual acuteness possible should be obtained.

Ordering of Glasses.—Glasses are ordered for astigmatic eyes according to the general rules already given. For distance, the full correction is ordered in myopic astigmatism and usually in mixed astigmatism, but taking into consideration the directions on pages 135 and 147; in compound hyperopic astigmatism the spheric lens is usually weakened to meet the requrements of accommodation, but the full cylindric lens should be ordered. In simple hyperopic astigmatism it may be necessary to add a concave spheric lens; thus, if the correction under full mydriasis at 4 meters should prove to be + 1.50 D cyl., axis 90°, the formula for the glass to be worn after return of accommodation would be — 0.25 D sph. ⊂ + 1.50 D cyl., axis 90°. In compound myopic astigmatism the spheric lens is sometimes weakened for near work.

At present there is no uniform plan for the designation of the meridians in astigmatism, and, consequently, formulas for glasses intended

[1] For full directions in regard to the use of the lens see article by E. Jackson Ophthalmic Record, August, 1907.

to correct astigmatism do not have a uniform meaning in all parts of the world. Drs. Thomson and Harlan[1] have conveniently summarized three systems as follows:

1. The zero is placed at the end of the horizontal meridian to the patient's left, and the degrees are counted on the upper semicircle to 180° at his right.

2. Zero is placed at the top of the vertical meridian, and the degrees are counted to the nasal and temporal sides to 90° at the horizontal meridian.

3. The zero mark is placed at the nasal extremity of the horizontal meridian in each eye, and the degrees are counted on the upper semicircle to 180° at the temporal extremity.

The first is the one in almost universal use in this country, the formula for the glasses being written in accordance with the markings on the trial-frame.

Irregular Astigmatism.—A low degree of this defect exists in nearly all eyes, but it does not interfere with good vision. When its degree is increased by irregularities of the corneal surface from keratitis and cicatrices, the vision is very much reduced, and where such lesions are extensive, optical therapeutics may be unavailing. Often, however, within the pupil space small areas may be found in which the refraction is tolerably uniform, and vision may be decidedly improved by lenses—spheric and cylindric. All such eyes should be carefully studied by objective methods, and full trial with lenses should be made. Stenopaic spectacles render vision more distinct, but they embarrass the wearer by limiting the field of vision. An iridectomy sometimes improves vision by displacing the pupil toward a more regular portion of the cornea.

Surgical Treatment of Astigmatism.—It has been proposed to correct astigmatism by incising the cornea with a Graefe knife, or by producing a wound two-thirds of the depth of the cornea with the galvanocautery (Laus). The operation should be performed on the meridian of greatest refraction (Borsch). The author has had no experience with these procedures.

Anisometropia.[2]—This term includes cases in which one eye is much more hyperopic or myopic than its fellow, or where one eye is astigmatic and the other not, or where myopia exists in one eye and hyperopia in the other. Anisometropia may be congenital or may be acquired. No general rule for the management of cases of this character can be given, but the author agrees with Duane that "in the majority of cases of anisometropia, even those in which the difference in refraction exceeds 2 D, the full correction can be applied

[1] Archives of Ophthalmology, 1893, vol. xxii, pp. 251–261. This paper contains an excellent discussion of this subject and an analysis of the arguments for the various systems.

[2] This term, according to Suker, is often inaccurately applied. He would employ it only to describe an unequal amount or degree of the same kind of refractive error in the two eyes. To describe a different kind of refraction in the two eyes he prefers the word *antimetropia*.

with success." The patient, however, must be required to wear the glasses constantly, and must be willing to bear with temporary discomfort while the eyes are becoming accustomed to the lenses. The causes which give rise to discomfort may be summarized as follows: Diplopia and asthenopia from the unequal prismatic effect of the unequally strong lenses; diplopia from imbalance of the ocular muscles, with the full correcting lenses the double images being more manifest; and difficult binocular vision because the retinal images of the two eyes are of a different size, a cause, however, which is considered fallacious by Duane. Exophoria and hyperphoria are often associated with anisometropia; squint may be caused by this refractive condition and may be materially improved by the use of the correcting lenses. If discomfort ensues, success may follow the attempt to train the function of the more defective eye by temporarily excluding the other from vision. Corrections by prisms of the hyperphoria is often of distinct advantage.

Presbyopia.—The accommodation diminishes gradually from early life onward, and the near-point recedes farther from the eye with each succeeding year. When by this recession the near-point reaches a distance of 30 to 40 cm. from normal eyes, it interferes with their use at close range, and convex lenses are usually required. *Presbyopia* is a physiologic condition and is a normal result of advancing years.

Causes.—The cause of presbyopia consists in loss of the elasticity of the crystalline lens, which is thus prevented from assuming the increased convexity which constitutes the essential factor of accommodation. This increase of convexity, necessary for seeing near objects, must be supplied to the eye by a suitable lens.

In emmetropic eyes at about the age of forty-five the near-point has generally receded beyond a comfortable or satisfactory reading distance. Unusual visual acuteness, or vigor of accommodation, however, may enable a person to dispense with glasses for several years longer. A visual acuteness of $\frac{6}{4}$ permits its possessor to see the same object distinctly at 30 cm., which another individual with a vision of only $\frac{6}{6}$ would have to hold at 20 cm. Patients occasionally postpone the time of wearing reading glasses by holding fine print in a bright light, the resulting contraction of the pupil rendering vision more distinct. Presbyopia must be distinguished from hyperopia, which is often latent and confounded with it. Correction of hyperopia restores the far-point of the eye to infinity.

Correction of Presbyopia.—In the first stages of presbyopia, while considerable accommodation still remains, a weak convex lens is required, which enables the person to see near objects by rendering the rays less divergent, as if they came from a somewhat greater distance.

There is still a range of vision from the focal distance of the glass to the near-point. A person who has an accommodation of 3 D, and requires + 1.50 D in addition, will have a range from the focal distance

of the glass $\frac{1 \text{ meter}}{1.50} = 66$ cm. to his near-point through the glass; 3 D + 1.50 D = 4.50 D; $\frac{1 \text{ meter}}{4.50} = 22$ cm.

When the accommodation is entirely obliterated at seventy-five years of age the convex glass must be stronger. The rays are now rendered parallel, as if they came from an infinite distance, and the object must be held at the focus of the lens. There is, therefore, no range of vision.

The presbyopic glass is estimated after the eye has been rendered emmetropic by neutralizing any hyperopia or astigmatism which may be present (for the management of myopia and myopic astigmatism in these circumstances see page 164).

The *near-point* of vision should be carefully determined for each eye separately. The ability to read 1-meter type at 30 cm. is not equivalent to the act of accommodating for 30 cm.; in order fairly to accommodate for 30 cm. the patient should be able to read type which represents normal vision at 30 cm. (see page 39). If the accommodation is normal, the near-point will correspond closely with the figures given in the table. The additional refractive power required may then be calculated. Unduly strong glasses should not be employed in approximating the near-point, lest the far-point be brought too close and serious discomfort ensue. Most persons read at an average distance of from 33 to 40 cm. and in early presbyopia considerable range of vision exists on either side of these points; but at sixty years and later there is little play—the near-point and far-point are close together. A glass with which the patient reads easily at 33 to 40 cm. may then be ordered unless visual acuteness is much diminished.

Table of the Position of Near-point at Different Ages

Age.	Accommodation.	Point.
45	3.50 diopters	29 cm.
50	2.50 "	40 "
55	1.75 "	57 "
60	1 "	100 "
65	0.50 "	200 "
70	0.25 "	400 "
75	00 "	∞ "

At the age of forty-five it is usually necessary to supply a + 1 D spheric lens for reading, provided the eye is emmetropic; if the eye is hyperopic, 1 D + the correction for the hyperopia; if myopia exists, + 1 D is not required. Plus 1 D added to the 3.50 D of accommodation which the eye possesses at forty-five years = 4.50 D; this brings p to 22 cm. $\left(\frac{100}{4.50} = 22\right)$, and r to 100 cm.

At fifty years of age + 2 D is usually required, with the same modifications in case of hyperopia or myopia. This glass, added to the accommodation which the eye possesses at 50—viz., 2.50 D—also

makes 4.50 D; this brings p to 22 cm., but r is now only 50 cm. distant. Indeed, in most circumstances a + 1.50 or 1.75 D is sufficient.

At fifty-five years + 2.50 D is the glass usually required, which, added to the accommodation (1.75), gives a refractive power of 4.25 D; p = 23.5 cm., r = 40 cm. If stronger lenses than this are used r is brought still closer, and the patient is forced to hold the book near his face. If $V = \frac{6}{6}$, it is not necessary to order a glass stronger than the one recorded; indeed, usually + 2.00 D or 2.25 D is sufficient, as most persons prefer a glass which enables them to read, resting the book on the lap or the arm of a chair. It is a great mistake to order presbyopic glasses which are stronger than the actual ocular requirements (see also page 162). These glasses are for emmetropic eyes. In hyperopia with presbyopia they are to be added to the hyperopic correction. Visual discomfort (asthenopia) often develops with the first attempts to use presbyopic glasses, owing to the development of temporary convergence —insufficiency from relaxation of accommodation. In these circumstances it is usually necessary to reduce the strength of the glass. In the presence of exophoria prisms base in may be added to the correcting glass and such combinations are often productive of highly satisfactory results (page 644). Exercises with prisms are also useful (page 643).

It is of the utmost importance carefully to correct astigmatism before adjusting presbyopic glasses, moreover astigmatism of low degree. Especially is this true in the early years of presbyopia, for example, between the forty-fifth and fiftieth years of life. The necessity of search for inverse astigmatism in these circumstances has been referred to (page 155).

As visual acuteness diminishes a stronger lens may be necessary to enable the object to be held closer, and thus subtend a larger visual angle. The glass may be increased to 4, 5, or even 6 D. The strong glasses necessitate the close approximation of the object and a corresponding diminution in the field of vision. The only rule in the selection of such glasses is to give that glass which affords the necessary vision with the least inconvenience. With very great diminution of sight, requiring glasses of 8 or 10 D, binocular vision is impossible, and the better eye should be supplied with a correcting glass, and the other excluded from vision.

With binocular vision, the reading glasses for the two eyes should be equal in strength; consequently, when a different degree of ametropia exists in the two eyes, a corresponding difference should be made in the reading glasses. Occasionally, in the absence of ametropia, or even after its correction, when present, there is an inequality of the accommodative power in the two eyes. Thus, a patient of fifty years may have 2.50 D of accommodation in the right eye, and only 1.50 D of accommodation in the left. Under such conditions it is usually necessary to order a correspondingly stronger reading glass for the eye with the weaker accommodation.

Frequently, modifications are required in the strength of the glass

to suit particular vocations—for example, reading music, reading in the pulpit, working at a bench, playing cards, etc. In these circumstances it is necessary to ascertain the distance from the eye at which the work is placed, and to order a glass whose focal distance is not less but, if possible, somewhat greater than the distance required. Not infrequently the patient must be provided with two sets of glasses—one pair for the ordinary reading distance and another of greater focal length for work at a longer range. Thus, a patient of fifty-five years may require + 2.25 D for reading, but for playing the piano + 1.25 D. This correction of accommodative defects at which may be called an *intermediate distance* is most important, and the character of the patient's work must always be carefully ascertained before ordering a glass.

In myopia, myopic astigmatism, and mixed astigmatism the rules for the selection of reading glasses require particular mention. Patients with low degrees of myopia, not higher than 2 D, do not need reading glasses at as early an age as emmetropic or hyperopic subjects. The amount of myopia may be considered the equivalent of the convex glass suitable for the correction of the presbyopia. A myopia of 1 D, consequently, would enable a person to attain the age of fifty without the necessity of reading glasses. At that age he would require + 1 D for reading, and at fifty-five + 1.50 D, and at sixty, possibly + 2 D, depending upon his visual acuteness. A myope of 2 D could dispense with reading glasses until the age of fifty-five (often until a later period); then he would require + 0.50 D; at sixty, possibly + 1 D. A myope of 3 or 4 D never becomes presbyopic in the ordinary sense; he can read at any age without glasses.

In higher degrees of myopia it is necessary to order a concave glass from 2 to 5 D less than the full correction. The age has little influence on the amount of reduction; myopes readily relax accommodation; the degree of myopia and the visual acuteness are the two important factors. A concave glass is given which will extend the far-point to a comfortable distance. A myope of 6 D would probably require from − 3 to − 4 D for reading; a myope of 10 D, about − 6 D, and a myope of 15 or 20 D would require a reduction of 5 to 6 D from the full correction. In these high grades vision is much reduced, print cannot be seen unless held close to the eye, so that extension of the reading distance is out of the question. The farthest point at which a book can be read should be determined, and a glass given of the same length of focus. Prisms are often necessary. When the vision is much reduced, myopes usually read with one eye without the aid of any glass.

A patient with simple myopic astigmatism generally should be provided with a convex cylinder of the same number, its axis being reversed. Thus, a patient whose myopic astigmatism is corrected by − 2 D cyl., axis 180°, will be comfortable with a + 2 D cyl., axis 90°. This glass produces a myopia of 2 D in all meridians, and gives the patient a comfortable reading range.

But if the degree of myopia thus produced is too great for comfortable reading a concave spheric lens may be added to the convex cylinder.

Thus, an astigmatic eye corrected by a − 4 D cyl., axis 180°, would probably require as a presbyopic correction − 1.50 D sph. ⌒ + 4 D cyl., axis 90°. Naturally, the strength of the concave sphere must be determined by the patient's age and visual requirements.

If the degree of astigmatism is unequal in the two eyes, a spheric lens is required over one eye to equalize the refraction. For example:

1. R. E. − 5 D cyl., axis 180°. L. E. − 3 D cyl., axis 180°. This case requires a − 2 spheric lens to be added to the right eye—viz., − 2 D sph. ⌒ + 5 D cyl., axis 90°, to make its refractive power equal to that of the left, + 3 D cyl., axis 90°.

2. R. E. − 1 D cyl., axis 180°. L. E. − 2.50 D cyl., axis 180°. In this instance, according to the circumstances, age, etc., one of the following combinations may be ordered: R. E. + 1 D cyl., axis 90°; L. E. − 1.50 D sph. ⌒ + 2.50 D cyl., axis 90°; or R. E. + 1.50 D sph. ⌒ + 1 D cyl., axis 90°; L. E. + 2.50 D cyl., axis 90°. Both of these combinations equalize the refraction of the two eyes, the first by producing in each eye a myopia of 1 D, the second, a myopia of 2.50 D.

In cases of compound myopic astigmatism, should the myopia amount to several diopters, the reading glass is secured by a sufficient reduction of the strength of the spheric without change of the cylindric lens.

If, in lower degrees of compound myopic astigmatism, it is desirable to increase the refraction one or more diopters, the procedure is somewhat different. Thus, if the combination is − 0.50 D sph. ⌒ − 1 D cyl., axis 180°, and the spheric lens is omitted, + 0.50 D is gained; by substituting for the concave cylinder a convex cylinder with its axis reversed, an additional gain of 1 D is secured; + 1 D cyl., axis 90°, in this case is equivalent to adding + 1.50 D sph. to the original combination. If still more refractive power is desirable—*e. g.*, + 2 D, + 0.50 D sph. ⌒ + 1 D cyl., axis 90°, gives the additional amount.

In another combination, − 0.75 D sph. ⌒ − 4 D cyl., axis 180°, it is desired to add + 2.50 D for reading. Dropping the − 0.75 D spheric lens, + 0.75 D of refractive power is obtained; substituting for the concave cylinder, convex 4 D cyl., axis 90°, + 4 D more are gained, making + 4.75 D. This is too high, hence it would be necessary to combine − 2.25 D sph. ⌒ + 4 D cyl., axis 90°, in order to obtain the desired + 2.50 D. A simpler method of procedure in this case would be to drop the − 0.75 D spheric lens; the uncorrected myopia would then furnish 0.75 D of the requisite 2.50 D, leaving 1.75 to be obtained. A + 1.75 D added to the − 4 D cyl., axis 180°, would make the proper combination.

In mixed astigmatism a combination of spheric lens and cylinder is usually employed, and by using a concave spheric and convex cylinder the combination necessary to produce any additional refractive power can easily be found.

If the myopia produced by the convex cylinder alone is greater than the power of the lens desired, a concave spheric lens equal to the difference may be given, thus: To the combination − 3 D sph. ⌒ + 5 D cyl., axis 90°, it is desirable to add + 2 D. − 3 + 2 = − 1, hence

− 1 D sph. ⌒ + 5 D cyl., axis 90°, is the glass required. Again, to − 1 D sph. ⌒ + 3 D cyl., axis 90°, it is desirable to add + 2.50 D. − 1 + 2.50 = + 1.50, hence + 1.50 D sph. ⌒ + 3 D cyl., axis 90°, is the necessary glass.

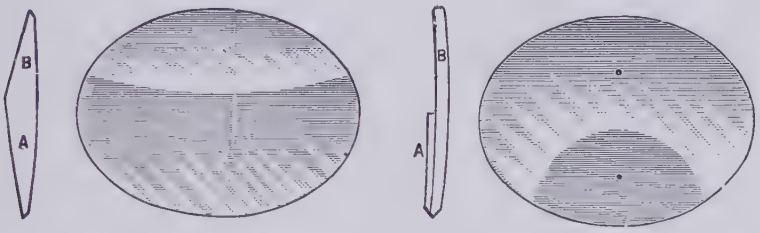
Distortion of Objects by Cylindric Lenses.—It is important, in ordering reading glasses containing cylindric lenses, to give attention to the relation of the axes of the cylindric lenses. It has been assumed, for the sake of simplicity, that the axes of convex cylinders are placed at 90° and the axes of concave cylinders at 180°. It is a frequent condition in astigmatism to have one principal meridian inclined 15° to the right of the vertical in one eye, while the meridian of the same refraction in the other eye is inclined the same amount to the left of the vertical. This produces no serious disturbance in wearing the glasses if they are properly centered, although at first a rectangular figure appears like a rhombus. In a little time the eyes adapt themselves to the glasses, and this appearance is lost.

When the meridians of similar refraction are at greater angles than this, especially if the cylindric lenses are strong, there is often inconvenience in wearing them on account of the prismatic deviation and the unequal distortion of objects which cylindric lenses produce. Occasionally the axes are as much as 90° apart, one at 45° and the other at 135°, or one at 90° and the other at 180°. The glasses now deviate rays from an object in different directions, according as the eye looks through the glasses above or below the optical centers, or to the right or left of them. Such a case would be represented by + 3 D cyl., axis 180°, in right eye, and + 3 cyl., axis 90°, in left eye. The difficulty is not obviated by ordering a formula like the following: R. + 3 D cyl., axis 180°, L. + 3 D sph. ⌒ − 3 D cyl., axis 180°, because the same displacement results. It will be found that the best solution of this difficulty is to ascertain the distance from the eye at which the person usually holds the book, and the relative position it occupies to the eye. The direction of the visual lines may thus be determined, and the optical centers of the glasses should be so placed that the visual lines will pass through them. There is then no deviation. Of course, this renders necessary a separate pair of glasses for reading. When cylindric lenses with axes in unusual directions are required for distance, the optical centers should bear the same relation to the visual lines in distant fixation. These disturbances are aggravated by removing the glass farther from the eye, and, conversely, the trouble diminishes as the glass is brought nearer to the eye.[1]

Bifocal Lenses.—When presbyopic patients require two sets of glasses, one for distance and one for reading and close work, it is the custom, instead of providing them with separate sets of glasses, prescribed according to the rules set forth in the preceding paragraphs, to order *bifocal lenses* (Fig. 81). By means of such glasses the inconve-

[1] Consult interesting papers in the Archives of Ophthalmology, vol. xviii, by Dr. J. A. Lippincott; in the Ophthalmic Record, vol. i, No. 1, by Dr. G. C. Savage. And Dr. R. J. Phillips, in the Annals of Ophthalmology, vol. ii, p. 31.

nience of changing spectacles is avoided, and, moreover, the patient's eyes are constantly adapted by proper lenses to close and long ranges. A spheric lens, suited to needs of accommodation, ground very thin, is cemented on the lower portion of the distance glass, usually upon its inner side. The size of the additional segment varies. Generally one $12\frac{1}{2}$ mm. in height and 22 mm. in length is sufficient. The shape of the supplementary lens varies. Commonly it is an oval; sometimes it



1. 2.

FIG. 81.—Bifocal lenses: 1, Solid bifocal lenses; 2, cemented bifocal lenses.

is made in the form of a circle, and sometimes it is dome shaped. Another form of bifocal lens is one in which, for the usual presbyopic segment, there is substituted a small lens 15 mm. in diameter, made of flint glass and sunk into the distance lens, which is made of crown glass. The increased refraction of the small lens depends upon the higher index of the flint glass. Its exposed surface is ground to the same curvature as that of the larger lens. Lenses of this character are usually known

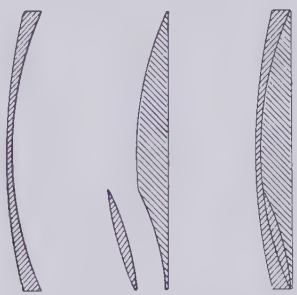


FIG. 82.—Borsch's bifocal lenses.

as "concealed" or "kryptok" bifocals. A "one-piece" bifocal lens may be ground out of one solid piece of hard crown glass, known in the trade as "Ultex one-piece bifocal."

SPECTACLES AND THEIR ADJUSTMENT

After the refraction of the eye has been determined and the proper combination of lenses selected, the glasses should be properly ground, mounted in spectacle-frames, and correctly adjusted to the patient's eyes. In place of spectacle-frames so called "eye-glasses" are much

employed; if they can be firmly adjusted, and the spherocylindric combination is not of high degree, there is no objection to their use, although they can never be as accurately applied as spectacles. Patients should not be allowed to wear glasses until the surgeon has satisfied himself that the formula for the lenses has been faithfully followed by the manufacturing optician.

In order to ascertain this he proceeds as follows: If a simple spheric lens has been ordered, this and a spheric lens from the trial-case, of the same number but opposite refractive character, are placed in close contact and some distant object observed through the combination, while the glasses at the same time are gently moved up and down and to and fro. If the glass is correct, this maneuver has no influence upon the size or position of the object, which appears exactly as it would if it had been looked at through a piece of plane glass. The glasses are then said to neutralize each other. If the lens ordered does not neutralize the test-glass from the trial-box, a weaker or stronger number is tried until the glass is found which produces complete or nearly complete neutralization. Thick bispheric lenses of different refractive character will not neutralize each other entirely, even if they are of the same number. The convex lens always preponderates. With a suitable "lens-measure" the character of a lens can be quickly determined.

If a cylindric lens has been ordered and has been correctly ground, it will be neutralized by a cylinder of the same number but of opposite refraction, with its axis turned to the same angle as that of the lens ordered. On shaking these two lenses, which are placed in contact, there should be no motion of the object viewed through them. The direction of the axis of a cylinder may be determined by finding the position in which the lens may be shaken without producing any motion of the object. For example, if the axis of the cylinder is vertical, no motion in the object looked at would occur when the spectacle lens is moved up and down. A line drawn on the glass with a pen marks this, and by placing the lens thus marked on a *protractor*, the degree of the angle may be read off. Alfred Cowan has designed a useful "axis finder," which is practically a transparent protractor which can be placed directly upon the lens to be tested.

A combination of spheric and cylindric lenses is to be tested by a spheric lens held on the spheric surface of the spectacle lens, and a cylindric lens held on the cylindric surface of the spectacle lens, and proceeding in the manner just described.

The *optical center* is ascertained as follows: The lens is held by its edges between the finger and thumb, and, care being taken not to hold it obliquely, it is passed from right to left until the test-object (a vertical line) forms a continuous line above the lens, through the lens, and below the lens. If the axis of the lens is not exactly in line with the test-object, the part seen above and below the lens will not coincide with the part seen through the lens. When a continuous line is obtained through the lens with the object above and below, the lens should be marked with a line drawn across its surface over the part where the line

is seen, just as the outline of a figure is traced on a transparent plate. The glass is now turned around so that the line is at right angles to its former position; another portion of the lens is found through which the test-object is also seen in a continuous line with the part above and below. This is traced on the glass with ink, and the intersection of the two lines thus traced marks one extremity of the axis of the lens. In most lenses the distance from the surface to the center is so slight that we may consider this point on the surface as the center, and each lens should have its center marked by a dot of ink. Strong lenses may be centered more easily by using the window-bars, while the glass is held close to them, or the edge of a card or sheet of paper, which is laid on the desk. Still greater accuracy may be obtained by using a card on which two lines are drawn, crossing each other at right angles; both principal meridians may in this way be found at once; the optical center then lies over the intersection of the lines.

The spectacles should next be placed on the patient, and the position of these centers in relation to the pupil carefully noted. The patient is first asked to look across the room; the centers of the pupils should correspond with the dots on the glasses. Next, the patient is required to look at the finger of the surgeon held at 40 cm. distance, and it will be noticed that the centers of the pupils and the dots no longer coincide, but that the former have passed to the inner side of the latter. If the glasses are for distance or for constant wear, the space between the centers of the lenses should be the same as the interpupillary distance; if the glasses are for reading alone, the distance between the centers must be lessened. The ordinary reading distance being 40 cm., the visual lines converge to this point, and the farther the glasses are from the center of rotation, the nearer the centers should come to each other; therefore it is necessary to make the distance between the centers of the reading glasses from 2 to 4 mm. less as compared with those of distance glasses, so that the visual lines may pass through these centers. Thus: The center of the pupil deviates inward about 1 mm. in fixing at a point 40 cm. distant, as the pupil is 11 mm. in front of the center of rotation; a glass placed 13 mm. in front of this would require its optical center to be 1 mm. farther inward than the pupil—2 mm. in all. The two centers should thus be 4 mm. nearer together in reading glasses than in those for distance.

Should glasses be ground with badly placed centers—that is, too far apart or too close together—the most unpleasant consequences may arise: obstinate diplopia, severe neuralgia, headache, and tendency to squint.

The patient should observe some distant object while the interpupillary distance is measured during distant fixation, but should fixate his eyes on the finger-tip of the observer, held about 30 cm. from his eyes while the measurement is made during convergence. There should be a variation of 2 mm. between these two measurements. If the difference is greater than this, there is a probability that the patient has an insufficiency of convergence, and, in this case, the centers of

convex glasses should be brought closer together; those of concave glasses placed farther apart. In order to ascertain the amount of deviation which is produced by decentering a spheric lens see page 20.

Reading glasses should be tilted forward and placed about 5 mm. lower than those for distance, in order to conform with the depression of the visual line in reading. Spectacles are always to be preferred; but the prejudice of many patients in regard to spectacles will often have to be respected. The tilting forward of eye-glasses is rather an advantage in reading, and in myopia the effect of this tilting is equivalent to a cylindric lens with a horizontal axis. This fact accounts for the preference shown by some patients for a simple concave spheric uncombined with a cylindric lens, in spite of the existence of a slight degree of astigmatism.

When separate glasses are required for distance and reading, it is often very inconvenient to make the change from one to the other. The two glasses may be combined in the same frame by making the lower half suitable for reading and the upper half for distant vision (Franklin or split bifocals). *Bifocal lenses*, as already described, constitute a more suitable arrangement (see page 166). "Hook fronts" are very convenient for making a rapid change from reading to distant vision, or "half-hook fronts" may be employed. Occasionally, for special purposes, *trifocal lenses* are manufactured—that is, an upper segment correcting the distant vision and a lower segment correcting the close vision are cemented in a lens which corrects the intermediate vision.

CHAPTER V

DISEASES OF THE EYELIDS

Congenital Anomalies.—Complete absence of the lids (*ablepharia totalis*) or their partial development (*ablepharia partialis*) is a rare anomaly. If the defect is of such a nature that the lids are wanting and the orbit divested of any covering for the globe, the condition is designated *lagophthalmos*, a name which also, and perhaps more properly, has been given to a contracted state of the eyelids preventing their closure, independent of any muscular paralysis.

Cryptophthalmos is a condition in which neither eyelid nor conjunctival sac is present, but the exterior integument passes in front of, and buries an eye more or less developed.



Fig. 83.—Coloboma palpebræ and anophthalmos.

Cleft eyelid (*coloboma palpebræ*) is a fissure, in appearance not unlike a harelip, which may be confined to the upper lid (its most common situation), but which also has been noted in the lower lid (when the upper lid of the same eye is always also involved), and even in the upper and lower lids on each side. The center of the cleft may contain an intervening membranous portion, either movable or pressed against the cornea (Fig. 83), or may be clear, so that the cornea fits exactly into it when the eyes are directed straight forward.

Coloboma of the eyelids is most frequently associated with harelip and cleft-palate; rarely with facial defects and other congenital anomalies in the eyeball. The deficiency may be remedied by a plastic operation.

Symblepharon, or a cohesion, either partial or complete, between the eyelid and the ball, and *ankyloblepharon*, or a union between the margins of the lids, are unusual congenital anomalies. Sometimes only the middle portions of the lid borders are attached by a filamentous band, or the outer angles of the lids adhere, and produce the defect known as *blepharophimosis*.

Ectropion, or eversion of the edges of the eyelids, is a rare condition usually accompanied by increased size of the eyeball. *Entropion*, or inversion of the edges of the eyes, which in slight degree is said to be normal before birth, has been found associated with *distichiasis*, or the development of supplementary in-curved eyelashes.

In *congenital distichiasis* Kuhnt demonstrated that the second row of lashes consists of fine hairs from the posterior part of the intermarginal area. Meibomian glands are wanting, and their places are

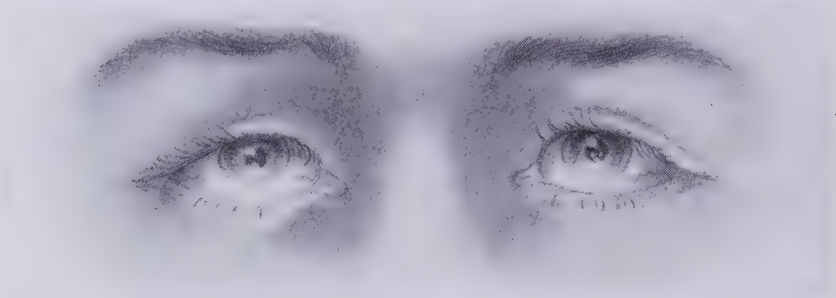


Fig. 84.—Congenital distichiasis and nebulous corneas (from a patient in the Philadelphia General Hospital).

taken by the abnormal cilia. Occasionally the condition appears to be hereditary.

The operations which are employed to rectify these conditions when of pathologic origin (see page 698) are suitable.



Fig. 85.—Epicanthus and congenital ptosis (from a patient in the Children's Hospital).

Epicanthus is a striking congenital anomaly giving rise to an apparent convergent strabismus, owing to the passage of a fold of skin from the inner end of the brow to the side of the nose, covering the internal canthus, its free concave border stretching outward. Thus the caruncle, lacrimal punctum, and, in aggravated forms, a considerable portion of the area of the lids, are hidden. Epicanthus generally is bilateral and is usually associated with ptosis (Fig. 85). The same condition in minor degrees is often seen in newborn children, and disappears with the subsequent development of the face and nose. A similar fold of skin at the outer commissure of the lids has been described, the so-called *external epicanthus*. True epicanthus may be hereditary, and the defect has been noted in several generations (von Hippel).

Epicanthus may be remedied by excising a portion of the redundant integument from the bridge of the nose, and stitching together the opposed surfaces.

Epitarsus is a somewhat wing-shaped duplicature of conjunctiva which passes from the fornix to be inserted near the lid border, and is so undermined that a small probe can be passed beneath it (Schrapringer). This anomaly is also described under the name *congenital pterygium.*

Congenital ptosis consists in a drooping of the upper lid over the eyeball. It may be unilateral or bilateral, the latter being the usual condition, but never amounts to complete closure. In one variety there is an actual redundancy of the lid tissue; in the other the lid is thin and the skin stretched, owing to imperfect development or absence of the levator palpebræ. It may be caused by a nuclear and, rarely, by a cortical lesion. The hereditary character of congenital ptosis is illustrated in H. H. Briggs' noteworthy report. Of 128 members of 23 families, 64 were affected with the ptosis—33 males and 30 females and one of unknown sex. His cases conformed to the Mendelian law of transmission.

This anomaly is often associated with other vices of conformation, especially epicanthus, and with paralysis of the exterior ocular muscles, especially the superior rectus, or this muscle may be absent; absence of the internal rectus (Lawford) and of both oblique muscles (Horles) has been recorded. Congenital ptosis may be corrected by one of the operations described on pages 691–695.

Congenital *fistula* of the upper lid has been reported. A *supernumerary eyelid,* presenting as a small growth at the inner canthus, has been described (J. L. Shoemaker and A. Alt).

Edema of the lids in general terms is *inflammatory* or *non-inflammatory* in origin. It is important to inspect the cornea and conjunctiva in all cases of lid edema; this is sometimes difficult and requires the aid of a lid-elevator. It may result from traumatism, the sting of an insect, contact with certain varieties of moths, notably the brown-tailed moth, and is seen with severe inflammations of the conjunctiva, of the cornea, the uveal tract, and especially with infections of the globe, *e. g.*, panophthalmitis and in association with acute glaucoma; also with hordeolum, acute chalazion and dacryocystitis. Associated with exophthalmos it is conspicuous in orbital cellulitis, tenonitis, and thrombosis of the cavernous sinus. Disease of the paranasal sinuses, especially of the ethmoid and antrum, is a frequent cause. It is a common symptom of general conditions (renal or cardiac) and sometimes occurs in a *fugitive,* and not infrequently *recurrent,* form. The last variety has been observed with migraine, at the time of the establishment of menstruation, and spontaneously without apparent cause. Some cases are analogous to urticaria. The eyelid is a common seat of *angioneurotic edema.* Some types of edema, non-traumatic in origin, have been called *essential edemas.* According to Trousseau, they are often *arthritic* in origin.

A condition has been described characterized by great symmetric swelling of both eyelids which present the usual appearances of chronic edema, and to which Sir Anderson Critchett has given the name *solid edema of the eyelids* (also called *Elephantiasis lymphangiodes*). The

affection has been regarded as a recurrent lymphangitis of lupoid origin (Morris). Not infrequently the patients have a history of repeated attacks of erysipelas (Eyre). Persistent, non-inflammatory edema of the lids is sometimes observed in children following measles (Lawson and Sutherland). In rare instances lid edema is a late symptom of syphilis.

Erythema of the lids appears in the form of a hyperemia, more or less diffused, under the influence of heat (sunburn), traumatism, and irritating poisons, or as symptomatic of a systemic disturbance.

A *passive hyperemia*, in which the superficial veins of the lids are dilated and the tissue red and slightly swollen, commonly is the result of prolonged bandaging of the eye, and is seen in an *active* state associated with most of the inflammatory diseases of the cornea and conjunctiva.

Urticaria, or **hives,** appears in the form of characteristic wheals associated with much tingling and burning sensation.

Treatment.—This consists in removal of the cause and the application of a soothing lotion—lead-water or extract of hamamelis.

Erysipelas rarely attacks the eyelids as a primary affection, but spreads to them from the contiguous facial area. The chief danger of the affection in this region is its liability to infect the tissues of the orbit, producing compression of the central vessels of the retina and blindness (see also page 661). It may spread to the membranes of the brain and be fatal. It is one of the causes of septic thrombosis of the cavernous sinuses (page 664). The characteristic red, shining, and later brawny swelling, and the formation of cutaneous vesicles and small abscesses, are the symptoms which establish a diagnosis.

Abscess of the lid (**phlegmon**) appears as a localized red elevation, and is often a severe form of furuncle or hordeolum. The entire lid is hyperemic and the conjunctiva injected and often edematous. There are much pain, headache, and fever. This affection is provoked by injury, exposure, and disease of the orbit, and sometimes arises without ascertainable cause, especially in debilitated people and children. In rare instances it has been followed by thrombosis of the orbital veins and cavernous sinus and has terminated fatally.

Treatment.—Pointing should be favored by hot, slightly carbolized fomentations or compresses soaked in boric acid solution. As soon as fluctuation is detected, or even earlier, a sharp knife may be thrust through the swelling, parallel to the muscle-fibers, and the contents evacuated; the cavity should be kept clean with an antiseptic fluid.

Furuncle of the lid is a localized inflammation of the skin and subcutaneous tissue, presenting symptoms analogous to abscess, which goes on to the formation of a central slough or "core." The surrounding and overlying tissue may become gangrenous in subjects of poor nutrition. The treatment does not differ from that of abscess of the lid.

Malignant pustule, or *specific anthrax*, caused by the entrance of the *Bacillus anthracis*, and *malignant edema*, or a form of *spreading*

gangrene, occasionally attack the eyelids. Anthrax usually arises among persons whose occupation brings them in contact with diseased animals or decayed animal matter. It occurs among wool-sorters, both in the brow and lid. Quite early a small papule surmounted by a vesicle appears; later a black central slough develops, surrounded by a ring of vesicles, and the whole area is red and brawny. Gangrene may follow an injury, influenza, the exanthemata, typhoid fever, whooping-cough, and erysipelas, but has also been described as an idiopathic affection. Sometimes gangrene of the lids is metastatic in origin and occurs during pyemia. *Noma of the eyelids*—that is, a symmetric gangrene of the lids and region of the lacrimal sac—has been described. Gangrene of the lids has been observed in diabetes.

Treatment.—According to the condition present, this should include incision, promotion of the separation of the sloughs by hot compresses steeped in boric acid solution, the use of the actual cautery to check the destructive tendency, and antiseptic lotions.

Usually staphylococci and streptococci are present, but in some of these gangrenous processes diphtheric bacilli have been found, and under such conditions serum therapy would be strongly indicated.

Blastomycosis of the Eyelids.—Blastomycotic dermatitis, which may affect any portion of the body, has involved the eyelids in about one-fourth of the cases thus far reported (Casey Wood). The affection begins as a red papule and gradually extends until it forms a flat, wart-like growth with a red elevated margin. Dry crusts are apt to cover its surface, and on their removal a bleeding rough surface is evident. Miliary abscesses are seen in the softened edges, from which and from the surface of the growth a mucopurulent discharge exudes. Although the conjunctiva may be injected, swollen, and granular, it is not involved further in the pathologic process. The disease is caused by an organism belonging to the genus *oïdium*, the spores of the fungus finding entrance owing to the injury of the skin surface. The affection may be mistaken for epithelioma, tuberculosis, or syphilis, and is differentiated by the clinical appearances and by an examination of the secretion from the miliary abscesses which will reveal the organisms. From the ocular standpoint the disease has been well described in this country by Casey A. Wood, W. H. Wilder, and Edward Jackson.

Treatment.—This should consist of excision of the diseased areas, the application of the *x*-rays and of radium, and the internal administration of large doses of iodid of potassium. Ectropion may occur and may need a plastic operation for its relief.

Hordeolum, or **stye,** consists of a localized, suppurating inflammation of the connective tissue in the margin of the lid or of one of the glands of the follicles of the cilia (Zeis' glands), and is almost always due to staphylococcus infection. This may remain a tender, circumscribed swelling, which becomes invested with a yellow cap, indicating suppuration, or it may cause considerable pain, with edematous swelling of the entire lid and chemosis of the conjunctiva. It is known by the name *hordeolum externum*, to distinguish it from a *hordeolum internum*,

which is the result of suppuration of a Meibomian gland. Some persons are subject to a mild type of styes which appear in the form of superficial pustules along the margin of the lid. A characteristic feature of hordeolum is its tendency to recur, and a single stye, or several at a time, may appear again and again for many weeks or months. Recurring hordeola may be the starting-points of a chronic blepharitis. Driving in the cold or dust and the strain of uncorrected ametropia predispose to this disorder. Frequent "attacks" of styes always indicate derangement of health, and are especially associated with constipation and menstrual irregularities. Girls about the age of puberty are commonly affected. Recurring styes may be one of the manifestations of focal infections in the nasopharynx, sinuses, teeth, tonsils, and intestinal tract.

Treatment.—A stye sometimes may be aborted by the vigorous application of a hot boric acid lotion or an ointment of the red or yellow oxid of mercury or applications of alcohol, 70 per cent. In the event of failure, suppuration should be encouraged by repeated applications of small compresses steeped in hot water, and on the earliest appearance of pus a deep incision should be made through the base of the swelling parallel to the edge of the lid. In persistent and recurring formation of styes treatment with bacterial vaccines achieves excellent results. A thorough general examination is necessary and treatment according to the findings.

Exanthematous eruptions on the eyelid are found during the course of the eruptive fevers. The pustules of smallpox, if they appear upon the eyelids, form by preference at the commissures, and in connection with the follicles of the eyelashes. The subsequent pitting from loss of tissue may cause considerable disfigurement.

Sometimes a pustule declines to heal and forms a chronic *post-variolous ulcer*. *Vaccine vesicles* (*vaccine blepharitis*) may form on the lid margins from accidental inoculation—*e. g.*, with the finger-nail previously in contact with a vaccine pox or vaccine virus. The vesicles may develop into a severe ulcer, and the bulbar conjunctiva and cornea may be involved.

Eczema of the lids, independently of that variety which is located upon the ciliary margin and which is one of the forms of blepharitis, may appear upon the general cutaneous surface of these structures, usually in association with its presence elsewhere on the face and csalp, and is seen in the *erythematous*, *vesicular*, and *pustular* varieties.

Eczematous eruptions upon the lids are also associated with inflammations of the cornea and conjunctiva and arise under the influence of prolonged bandaging. Atropin, when it produces conjunctivitis (see page 250), may cause an eczema of the lids and surrounding face.

Treatment.—This depends upon the character of the eruption. If this is vesicular, a useful application is a drying powder composed of starch, oxid of zinc, and camphor; if crusts have formed, these should be removed with as little bleeding as possible and with the aid of an alkaline solution, maceration of the epidermis being avoided, and one

of the following ointments employed: Plain oxid of zinc, or equal parts of oxid of zinc and vaselin to which 20 grains (1.3 gm.) of calomel to the ounce have been added; or subnitrate of bismuth in an ointment. Itching is relieved by the application of *lotio nigra* followed by zinc ointment. If the disease assumes a chronic type, some preparation of tar (*pix liquida* or *oil of cade*) may be used. Good results follow the use of aristol ointment, both in subacute and chronic cases.

As constitutional remedies, quinin, iron, and strychnin are recommended, and arsenic if the type is chronic. Proper regulation of diet, an occasional saline laxative, and good hygiene are important measures.

Herpes zoster ophthalmicus is a specific infectious and possibly contagious exanthem (Van Harlingen) characterized by an eruption of vesicles, situated upon inflamed bases, over the area supplied by two of the three branches of the ophthalmic, or first division of the trigeminus—viz., the frontal, through its supra-orbital and supra-trochlear branches, and more rarely the nasal nerve.

Neuralgic pain, heat, and redness of the skin precede the vesicles, which, varying in size from a pin's head to a split pea, appear in distinct crops or coalesce in irregular patches. At first they contain a clear yellow fluid, later becoming turbid, until at the end of a week or more they dry up, and the brown scabs drop off, leaving beneath decided and often disfiguring scars.

The disease may be mistaken for erysipelas, from which it should be distinguished by the acute neuralgic pain and the formation of the vesicles in the course of a given set of nerves.

Serious involvement of the eye itself, by the formation of blebs upon the cornea (*herpes zoster corneæ*), and by inflammation of the iris and ciliary body, is often associated with the disorder. Depigmentation of the iris (*vitiligo iridis*) may be a sequel of the iritic involvement (A. Lowenstein). More or less conjunctivitis is always present. The blebs on the cornea rupture and form ulcers, which leave permanent scars, and the iritis and cyclitis may pass on to a destructive inflammation of the deeper coats of the eye (ophthalmitis). Deep keratitis (*keratitis profunda*) may arise in connection with the herpes zoster, and a form of parenchymatous keratitis which precedes by several days the cutaneous lesions has been reported (Terrien). Herpetic eruptions on the sclera may occur during the course of herpes zoster in the form of small reddish nodules. Atrophy of the optic nerves and paralysis of the oculomotor and of the superior oblique have followed ophthalmic herpes.

Although the intra-ocular tension may be reduced in this disease, *acute glaucoma* and *glaucoma secondary* to cyclitis are complications which have been observed in a number of cases. Weeks attributes elevated intra-ocular tension to changes in the aqueous humor, filtration being checked on account of increase in albuminoid and globulin substances. In the author's experience in most of the cases the rise of tension has occurred in connection with, or as the result of, cyclitis.

Inflammation of the tissues of the eye is most apt to occur when the nasociliary branch is affected, and the vesicles extend to the tip of the

nose, because from this branch, through the lenticular ganglion, arise the nerves supplying the iris, ciliary body, and choroid. This is not an invariable rule, and destructive disease of the eyeball may appear even when the nasal branch is not involved. A severe and most intractable neuralgia often remains after the subsidence of the eruption.

Herpes zoster ophthalmicus is more frequently seen among elderly people of feeble nutrition than among adults and young children, but the latter may be attacked even in the absence of constitutional depression. During the war the author observed an unusual number of cases of herpes zoster among young adults otherwise in good health. Some of these cases represented complications following antityphoid inoculations (see also page 361). Herpes zoster has been attributed to an affection of the gasserian ganglion; in some instances a relation between this disease and focal infections has been suggested.

Treatment.—The disease runs an acute course and tends to spontaneous recovery in two or three weeks. Locally, anodynes are useful —lead-water and laudanum, weak carbolic acid lotions, and preparations of belladonna. Ichthyol ointment is valuable. Desiccating powders (rice-starch, calomel, zinc oxid) are useful. Severe pain must be mitigated by opiates and morphin hypodermically, while the best constitutional remedies are full doses of quinin and iron, and later arsenic. Salicylate of sodium and acetyl-salicylic acid (aspirin) are valuable, and McNab recommends ionic medication, with sulphate of quinin by means of the positive pole over the affected area. The postneuralgic pain may be relieved by croton chloral hydrate in doses of 5 to 10 grains (0.324–6.5 gm.) every four hours, and by the use of a mild galvanic current. If conjunctivitis, keratitis, iritis, or cyclitis arises, it requires the treatment directed to the relief of such conditions, which is detailed in the special sections devoted to their consideration (see page 278). Should glaucoma arise the treatment must be modified according to the conditions, in some cases with cyclitis and rise of tension the cautious use of a mydriatic (scopolamin or homatropin) has achieved good results.

Herpes Febrilis of the Lids.—Occasionally one or several groups of herpes vesicles develop upon the eyelids. The lesions usually appear in the form of a small cluster or a coalescent patch. They may arise during the course of acute infections, for example, influenza, and in association with disorders of the respiratory tracts. Vesicles may also form on the cornea. The lid is swollen, reddened, and the disease gives rise to a burning and itching sensation. The lower lid is affected more commonly than the upper (Fig. 86).

Treatment.—The best application is ichthyol ointment, and under its influences the lesions rapidly disappear. The associated conjunctivitis should be treated with the usual applications—boric acid and argyrol.

Blepharitis is the term applied to the various grades of subacute and chronic inflammation of the border of the eyelid, which, for clinical purposes, may be gathered into two groups—*non-ulcerative* and *ulcerative blepharitis*. The former may be studied under several subdivisions:

1. **Hyperemia of the Lid Border** (*Hyperæmia Marginalis; Vasomotor Blepharitis*).—The margins of the lids have an unpleasant slightly swollen, red appearance. Exposure to cold wind or any strain upon the accommodation causes a feeling of heat, followed by burning and lacrimation. The redness is caused by the passive congestion of the superficial blood-vessels. Scales or crusts are absent or but sparingly present.

2. **Simple Blepharitis** (*Seborrhea of the Lid Border; Blepharitis Ciliaris; Squamous Blepharitis*).—This variety depends upon an abnormal secretion of the sebaceous glands, and results in the formation of scales and crusts situated on the margin of the lids at the bases of the eyelashes, or adhering to them, and may appear in either a dry or a moist form. Removal of the hardened sebum exposes the skin, shining, red, and occasionally abraded. There is usually slight conjunctivitis. An accompanying seborrhea of the eyebrows and scalp may be present; both lids are invariably affected, and the patients complain of burning, inability to perform close work, and some dread of light. Occasionally, as the result of excessive secretion of the sebaceous glands, the lid borders are covered with yellow crusts, which in appearance have been compared to wax.

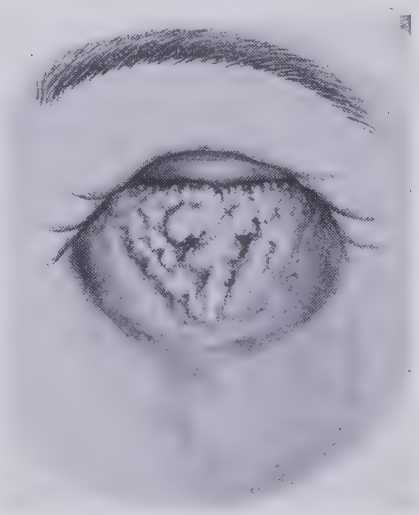


Fig. 86.—Extensive coalescing herpes confined to the lower lid.

Exposure to cold and dust and the use of the eyes quickly increase the congestion of the lids. If the disease is of long duration or is subject to frequent relapses, considerable thickening of the lid margins is evident, due to the inflammation surrounding the glands in the skin and tarsus. Crust formation on the lid margins due to a deposit of dried conjunctival secretion may simulate blepharitis. In these circumstances removal of the scales will demonstrate that the underlying skin is normal.

The second, or *ulcerative*, form of blepharitis appears in several grades of severity as a special localization of—

Eczema Upon the Lid Border (*Blepharitis Ciliaris; Blepharitis Ulcerosa; Psorophthalmia; Lippitudo Ulcerosa; Tinea Tarsi; Sycosis Tarsi; Ophthalmia Tarsi*, etc.).

(*a*) *Superficial Form* (*Marginal Eczema*).—This resembles in general that variety which has been described as hyperemia of the ciliary margin. The patient suffers from "weak eyes" and from frequent attacks of redness and soreness of the borders of the lids, associated with the

formation of crusts, small pustules, and ulcers at the roots of the lashes, without, however, seriously interfering with their nutrition or growth.

(*b*) *Solitary Form* (*Blepharo-adenitis Ciliaris, a name given by Arlt*).—This is characterized by the appearance of a circumscribed area of thickening and redness of the lid margin, upon which the cilia are matted together at their bases by the formation of thick yellow crusts. A single tuft of this kind may be present, or several on one lid border; the process is frequently unilateral, in this respect being unlike the squamous forms, which are bilateral. Removal of the crusts evacuates a few drops of thin pus from the surface of the ulcer which lies beneath, and the cilia, which usually come away with the scab, have swollen and thickened roots. Spots of eczema at the nares and in the hair of the scalp may be present, as well as disease of the lacrimal passages.

(*c*) *Pustular Form* (*Blepharitis Ciliaris Ulcerosa*).—This manifests itself as an eczema of the lid margins, in its worst types involving the four ciliary borders. Thick yellow crusts, which mat the eyelashes, form along the palpebral margins, covering deep ulcers which readily bleed, and which, often crater shaped, pass inward to the tarsus.

The inflammatory process, if unchecked, seriously interferes with the nutrition of the lashes and the edges of the eyelid. The former become stunted, curled, misplaced (*trichiasis*), or drop out, and may be entirely absent (*madarosis, tylosis*). The latter assume a rounded shape, are swollen, reddened, thickened, slightly everted, and deprived of cilia (*lippitudo*, or "blear eye," *hypertrophic blepharitis*), and if the punctum lacrimale is displaced or closed, an overflow of tears adds to the discomfort of the patient.

It is not always possible thus sharply to separate the various types of blepharitis, as they often shade one into the other; nor is it always safe to decide between those which arise from glandular hypersecretion and those which are due to eczema. After the cure of an ulcerative variety, small scales may form resembling the simple or squamous type, while the latter may also lead to, or be associated with, ulcerations.

Terson suggests a classification of affections of the lid margins from the dermatologic standpoint. He would distinguish two main groups, the suppurative and the squamous. The former, for the most part, includes affections of the hair-follicles and of the surrounding tissue—that is to say, either a *folliculitis* or a *parafolliculitis*. He regards ulcerative blepharitis as a process analogous to sycosis. The squamous form of blepharitis he classifies with seborrhea.

Etiology.—In the majority of instances blepharitis is a disease of childhood, and is common near the age of puberty; the aggravated forms, especially those resulting in chronic changes in the ciliary margins, are frequently seen in adults as the results of neglect. The malady may follow in the wake of an exanthem, particularly measles, and finds many subjects among anemic children of strumous or tuberculous habit, with blond hair and pale complexion. It is frequently associated with, and caused by, chronic conjunctivitis, even trachoma and phlyctenular conjunctivitis. The usual presence of ametropia has led to the

belief that this causes blepharitis (Roosa). There is no doubt that it aggravates and fosters the condition. In some families blepharitis is hereditary (Fuchs).

Of considerable importance in the origin of this affection are inflammations of the tear-sac, stricture of the nasal duct, and obstructive disease of the posterior nares, although it may be difficult, in individual cases, to decide whether the blepharitis has caused the closure of the lacrimal passages, or whether this has developed the blepharitis. Blepharitis is aggravated and excited by exposure to wind, dust, and heat, and by intemperate habits and loss of sleep. Finally, some instances appear to arise from an abnormal and probably congenital shortness of the lids (*microblepharon*), resulting in their insufficient closure during sleep (Fuchs).

Staphylococci are found in the pustules. McNab has frequently discovered the Morax-Axenfeld bacillus in some varieties of marginal blepharitis. Stubborn varieties may depend upon eczema seborrhoicum of the face; rarely the trichophyton fungus is found (*blepharitis trichophytica* of Mibelli). According to Raehlmann, the Demodex folliculorum may cause the disease (*blepharitis acaria*). It is, however, a not uncommon inhabitant of the normal eyelid. *Favus*, in the form of dirty, yellowish-white crusts, occasionally appears upon the eyelids, and may be mistaken for blepharitis. Microscopic examination of the crusts would reveal the mycelium and the conidia. The invasion of the follicles of the eyelashes by the *Trichophyton tonsurans*, when the brow or beard is similarly affected, produces an appearance closely resembling severe blepharitis.

Treatment.—This differs with the type of the disease, but in all cases the refraction of the eye should be ascertained and any anomalous condition corrected by suitable glasses. This will often cure an ordinary hyperemia of the lid margin, but if it is not sufficient, in addition to soothing lotions, the daily use of a douche of water at a temperature of 68° F., to which is added a little *eau de cologne* or alcohol, is serviceable. Stimulating salves do not yield good results in this variety, but the edges of the lids may be anointed with almond oil or vaselin.

In the cases classified among the seborrheas all crusts and scales should be removed by means of alkaline solutions—bicarbonate or biborate of soda, gr. viij to f℥j (0.52 gm. to 30 c.c.)—or with a 5 per cent. solution of chloral (Gradle), and one of the following ointments applied once or twice daily: yellow oxid of mercury, gr. j to ℥j (0.065–3.885 gm.), zinc ointment, or the salve advised by Gradle (milk of sulphur and resorcin, 3 per cent.).

Great care must be exercised to remove the crusts either with the aid of the lotions which have been mentioned or, after softening, with forceps, before the application of any salve. A satisfactory method is daily gentle scrubbing of the lid margins with the lather of a good neutral soap. For this purpose special soaps (ophthalmic soaps) may be employed. Crusts and scales may also be removed by means of a cotton-wound applicator which has been dipped in peroxid of hydrogen.

Yellow oxid of mercury salve or diluted citrine ointment or ichthyol (2–10 per cent. of the ammoniacal salt) may be applied to the lid margins.

In chronic cases all loose cilia should be extracted with epilating forceps, and any deep ulcers should be touched with the point of a crayon of nitrate of silver, or penciled with a solution of the same drug, or treated with a mixture of corrosive sublimate in glycerin (1 : 100 to 1 : 30 —Despagnet). In severe forms, or when it is desirable to try other remedies, diachylon ointment, 15 to 240 grains (0.972–15.5 gm.) of vaselin, boric acid ointment, 10 to 100 grains (0.65–6.5 gm.), or aristol ointment, 15 to 150 grains (0.972–9.72 gm.), will be found useful. Fridenberg recommends expression of the lid margins in order to remove the pathologic secretion from the glands and ducts. Picric acid (0.8 per cent.) in glycerin (Fage), and sulphate of zinc, gr. ij to f℥j (0.13 gm. to 30 c.c.), if the Morax-Axenfeld bacillus is present, serve a useful purpose. Stubborn blepharitis has been successfully treated by *ionic medication*, after the manner of Wirtz. A ½ per cent. solution of sulphate of zinc is employed; special electrodes are required.[1]

If the lacrimal passages are obstructed, they must be rendered patulous, and in all cases the anterior and posterior nares should be explored for disease. Septic conditions in the mouth (teeth, etc.) should be eliminated and infected tonsils removed.

The constitutional remedies include iron, quinin, and, if struma is present, cod-liver oil and lactophosphate of lime, with iodid of iron or syrup of hydriodic acid.

Blepharitis may be a mild affection and yield readily to treatment; or it may be stubborn, and require constant attention and frequent change in local measures to prevent deformities in the lid margins.

Phthiriasis (**blepharitis pediculosa**) occurs when the pediculus pubis or crab-louse forsakes its seat of predilection and finds a habitat among the eyelashes. The cilia appear sprinkled with a fine dark powder—the eggs of the parasites—which are usually found partially buried, head foremost, in the hair-follicles. There are some itching and redness. The affection in most instances has been observed in children. The lice may be removed by the application of blue ointment or a careful penciling with a bichlorid of mercury solution (1 : 1000).

Sporotrichosis of the Eyelids.—This disease may attack the eyelids in the form of a dermic granuloma with swelling of the preauricular, submaxillary, and cervical lymphatic glands (H. Gifford, Morax). Ulceration of the lid border and small abscesses of the lid skin may arise. Syphilis, tuberculosis, and blastomycosis must be excluded by cultivating the germ on maltose agar. The *Sporothrix beurmanni* is the fungus which usually is the active agent.

The *treatment* consists in local antisepsis and the administration of iodid of potassium.

Syphilis of the Eyelids.—Syphilitic affections of the eyelids exist either as a primary sore or as secondary or hereditary manifestations.

[1] For details consult Klin. Monatsbl. f. Augenheilk., Nov.–Dec., 1908; also Ophthalmoscope, Jan., 1911, p. 18.

A chancre usually appears on the area included by the lid borders and inner canthus, the tarsal conjunctiva and the culdesac, and therefore includes the lid border as well as the conjunctiva (de Beck). The lesion, generally on one lid, but in rare instances bilateral, begins as a pimple, which gradually develops into a characteristic, somewhat saucer-shaped ulceration, with rather rounded edges and indurated base. The lymph-glands in front of the ear and at the angle of the jaw are enlarged. Contagion has often occurred as the result of an application of the lips or tongue of an individual suffering from mucous patches in the mouth—as, for instance, in the act of kissing; or by the filthy practice of attempting to remove a foreign body with the tip of the tongue. Soiled fingers have also carried the contagion.

It is possible to mistake the affection for a stye, suppurating chalazion, ulcerated tear-sac, vaccine ulcer, or small rodent ulcer. In doubtful lesions a search for the *Spirochæta pallida* should be made; the Wassermann test should be applied.

Treatment.—Locally, the ulcer may be dressed with black or yellow wash. As soon as the diagnosis is established, the ordinary antisyphilitic remedies should be exhibited, especially salvarsan or neosalvarsan or the equivalent—arsphenamin.

The lesions of *secondary syphilis* upon the eyelids require no special description.

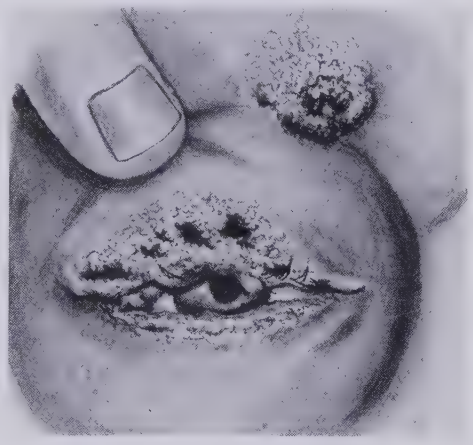


Fig. 87.—Extensive ulcerating gumma of the upper and lower lid of the right eye.

Among the later manifestations *gummas* of the skin of the lid, which break down into ulcers—so-called *tertiary ulcers*—are described. The lesions depicted in Fig. 87 disappeared under the influence of mercury and neosalvarsan.

A papular eruption may appear upon the eyelids of children the subjects of hereditary syphilis shortly after birth. A form of *blepharitis*, characterized by sharply ulcerated spots, has been described as the result of hereditary syphilis, and in subjects of this disease absence and falling out of the eyelashes have been noted and *rhagades* at the angles of the lids analogous to those at the angles of the mouth (Hutchinson). The latter condition also arises during secondary syphilis. A true *syphilitic blepharitis* in acquired syphilis has been recorded (Chaillous and Guéneau).

Tumors and Hypertrophies.—A variety of growths, cystic and solid, are found upon the eyelid and its border. Among the latter, *warts* or *papillomas* are common. These are benign, except when in elderly

persons, through irritation, they may take on an epitheliomatous nature. Although they may be removed by excision, many warts, especially those on the lids of old people, are more satisfactorily treated by applications of radium. In place of the ordinary elevated wart (*verruca*) a flat variety of the growth, which in persons of mature years may be pigmented, often develops (*keratosis senilis pigmentosus*). Superficial wart-like processes situated upon the intermarginal area of the lids have been described by Birch-Hirschfeld (*acanthosis nigricans*).

Small clear *cysts*, arising from Moll's glands, are common along the ciliary margin, often giving rise to considerable irritation. They should be punctured.

A reddish, wart-like mass may occur at the mouth of a Meibomian gland-duct. It should be excised.

Angiomas (*nevi*) are usually congenital growths, and exist either as bright red spots (*capillary angiomas*) or in the form of elevated, bluish, somewhat lobulated, *cavernous* growths, which may assume large proportions and extend from the lid to the forehead and temple. These cavernomas become turgid, purplish in color, and apparently increase in size if the child cries. They should be dealt with early in their existence, lest they spread into the orbit. In a patient under the author's care a tumor of this character extended to the apex of the orbit and involved the lacrimal gland. A tumor occupying the upper surface of the tarsus, soft in consistence and bluish in color, has been noted in the eyelids of babies. Clinically, it may be mistaken for a cavernoma, but examination after removal, which is usually accomplished without difficulty by an ordinary dissection, shows, as in Arnold Knapp's specimen, that it is composed of open spaces containing blood, which are lined with large endothelial cells, similar cells occupying the intervening regions. Angiomas arising from the lymphatics are known as *lymphangiomas;* those which develop from the blood-vessels, as *hemangiomas*. *Nevus pigmentosus*, and moreover, as the starting-point of a malignant growth, has been observed on the eyelids.

That operative interference should be practised which promises the least subsequent deformity of the lid. If they are small, capillary angiomas may be excised or cauterized with nitric acid; if of a larger variety, their blood-vessel structure may be destroyed with galvanocautery needles; or *electrolysis* may be tried, three gold-plated needles attached to the negative pole being inserted in various positions in the nevoid tissue, while the positive pole is attached to some distant point—for example, the arm. The *séance* should last from ten minutes to half an hour, according to circumstances. The application of carbon-dioxid snow has been efficient in the treatment of some angiomas. Radium has also been tried. It is possible sometimes to excise large cavernous angiomas, and if there is not sufficient skin to cover the defect immediately, to accomplish this subsequently by skin-grafting. Indeed, excision by an ordinary dissection, if it can be accomplished without serious loss of tissue, is a desirable method of treatment. In infants, however, the danger of shock and hemorrhage is great.

Occasionally ulceration occurs in an angioma and is followed by serious hemorrhage.

A disease characterized by an increase in the volume of the skin of the lid, which becomes folded and falls over its margin, but appears atrophic and may be transiently red, like the color of the cheek, has been described by Rohmer and others, and to it the name *angiomegaly* has been given. It symmetrically affects the upper lids, and has been attributed to a structural or functional anomaly of the vascular system.

Cutaneous horns (*fibroma; molluscum fibrosum*) occur as connective-tissue new growths, either sessile or pedunculated, sometimes associated with numerous similar tumors elsewhere on the body. A *cornu cutaneum* may grow from the margin of the lid (Fig. 88).

Neuromas of the plexiform variety occur on the eyelids, and *neurofibromatosis* accompanied by optic neuritis has been observed; also with hydrophthalmos on the corresponding side. *Lipomas*, which are probably extensions from the orbit, are benign growths which may be removed by careful dissection. *Ptosis lipomatosis*, at one time attributed to an accumulation of fat in the connective tissue of the upper lid, causing it to droop and its covering fold to hang over the palpebral border, is due to relaxation of the fascia connecting the skin with the tendon of the levator. *Fat hernias* of the upper lid have been described by Schmidt-Rimpler as the result of a congenital extension of the orbital fat through a defect in the orbicularis muscle. Occasionally, symmetric swellings of the lids, giving the impression of lobulation, appear toward their outer margins, similar to those noted at inner part of the upper lid in elderly people. They are probably due to pushing forward of orbital fat (Parsons). The baggy bulging of the lower lids in old people has a similar origin.

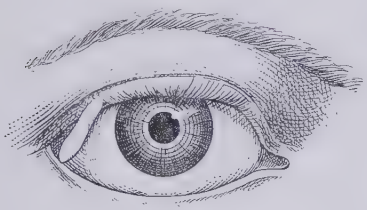


Fig. 88.—Cornu cutaneum of the upper eyelid (from a patient in the Jefferson Medical College Hospital).



Fig. 89.—Neuroma of the right upper eyelid and adjacent temporal region (from a patient in the Philadelphia General Hospital).

Uncommon forms of benign tumors are *adenoma* of the sweat-glands and their follicles, *adenoma* of the Meibomian glands (Knapp), *papilloma* of the ciliary border, *enchondroma* of the tarsus, and *myoma* of

the orbicularis (Schnaudigel). Hypertrophy and ossification of the tarsus have been reported (Herbert). An exceedingly rare condition of the eyelid is the so-called *sarcoid*, one case having been reported by Derby and Verhoeff. It begins as a redness of the skin and subsides, leaving one or more circumscribed infiltrated areas, or it may appear in the form of a small nodule.

Xanthelasma (*xanthoma*) is a connective-tissue new growth, with fatty degeneration, usually seen in the form of narrow, semicircular patches, most common upon the upper eyelids, although all four lids may be affected; it is more frequent in women than in men. The patches are yellow or buff colored, and on a level with the surrounding skin or slightly raised above it.

Excision, if this may be performed without producing ectropion, is the simplest method of treatment, but often yields unsatisfactory results, inasmuch as the xanthelasma reappears in the region of the excision. Electrolysis has also been recommended and often produces good results. High-frequency currents applied to the plaques by means of a special electrode are advised by Bordier. The application of trichloracetic acid to small xanthelasma patches has been commended, and the application of radium has been recommended by Schindler.

Chalazion (*Meibomian Cyst; Tarsal Tumor*).—This is a small tumor due to an inflammation of a Meibomian gland and the tissue which surrounds it. The growth begins by retention of the secretion of the Meibomian gland, which is a modified sebaceons gland, followed by a periadenitis and destruction of the tarsal cartilage, with passage of the tumor toward the conjunctiva (*internal chalazion*) or to the skin (*external chalazion*). Usually the process is a *chronic* one; sometimes it assumes an *acute* nature and there is inflammatory reaction (see also *hordeolum internum*). A chalazion may form in the excretory duct of a Meibomian gland, and then projects in a nipple-like body from the edge of the lid. Chalazia may be single or multiple, and in severe cases recurrences may be frequent until a chronic infection of the Meibomian glands and alteration of the tarsal cartilages take place. To this condition Weymann has given the name *tarsadenitis meibomica*. In association with nasal ozena an affection of the Meibomian glands has been described in which they become chronically inflamed, and pus, containing the ozena bacillus, exudes from their ducts. Pathologic changes in the Meibomian glands may be responsible for blepharoconjunctivitis. This relationship has been well studied by R. Sanford Gifford. A common condition, he finds, is one concerned with an excessive secretion of the glands; frothy material gathers at the angles of lids. *Concretions* and *infarcts* may be found in the glands. The latter appear as yellow spots beneath the tarsal conjunctiva.

Cause.—The cause of chalazia is not known, although Deyl and Hála maintain that they represent an infectious, bacterial process, the active bacilli being identical with xerosis bacilli. It is possible that some form of micro-organism is responsible for chalazia, which differs from the ordinary pus-producing cocci in that the chronic inflammation

which arises is not pus, but granulation tissue. But Sanford Gifford's bacteriologic studies revealed the xerosis bacillus in 50 per cent. of the cases; the staphylococci practically always; rarely the pneumococcus. It is possible, he thinks, that these bacteria may determine the type—staphylococci the acute, and xerosis bacilli the chronic, varieties. Chalazia may be associated with inflammation of the border of the lid and stoppage of the duct of the gland. Individuals affected with these growths not infrequently have ametropic eyes, especially where there is a tendency to recurrence in crops, like styes. Chalazia may be caused by chronic conjunctivitis. They are more common in adolescence than in youth, childhood, or in old age.

Symptoms.—The tumor grows slowly, unless it is of the acute type, and forms a firm swelling attached to the tarsus. The skin usually is freely movable over it; on the conjunctival surface a discolored, slightly protruding patch marks its position. Suppuration may take place in the growth.

A so-called acute chalazion may be mistaken for an external stye, from which it is to be distinguished by the more circumscribed character of the inflammation, and by the fact that the stye points in the edge of the lid; and a chronic chalazion for a sebaceous cyst from which it may be differentiated by the firmness of its attachment to the tarsus. A chalazion, a small sarcoma of the lid, and even a beginning glandular carcinoma have been confounded.

Pathologic Anatomy.—A microscopic examination reveals a collection of cells, the majority of which are of the small round variety, having their origin in the acini of the Meibomian glands. Sometimes large multinuclear (giant-) cells are evident, though inoculation experiments have shown that these are not tuberculous in type. The central part of the growth later undergoes a mucoid or colloid degeneration, and a cavity appears, which is filled with a cloudy fluid. There is no true capsule, and there are, consequently, no characteristics of a true cyst. *Retention cysts of the Meibomian glands* do occur, but, as Fuchs points out, they are essentially different from chalazia.

Treatment.—An ointment—2 grains (0.13 gm.) yellow oxid of mercury to 1 dram (3.9 gm.) of vaselin or lanolin—persistently rubbed into the skin over a chalazion will occasionally cause it to disappear, but almost always it is necessary to remove it according to the methods described on page 690. The eyes of patients who suffer from chalazia are usually ametropic and suitable glasses should be adjusted.

The malignant growths which appear upon the eyelids are *sarcoma*, *cylindroma*, *carcinoma* in the form of *epithelioma* or of *rodent ulcer* and *lupus*.

Sarcoma occurs as a primary tumor in both upper and lower lids, about 109 cases being on record (up to 1913), and usually is seen in children. In this country the recorded cases have been analyzed by Wilmer, Veasey, Alling, Friedenwald, Shumway, and the author. In Veasey's list, the youngest subject of lid sarcoma was seven months old and the oldest seventy-six years. The author has removed sarcoma

from the lid of a negress aged eighty-one (Fig. 90) and a hemangiosarcoma from the lid of a child aged five months. At first the growth is slightly elastic, and the skin moves over it freely, but the tendency is to rapid growth, ulceration, and involvement of the orbit. The tumor may attain large proportions. In a patient under the care of J. Chalmers DaCosta and the author the weight of the neoplasm was 247 grams. The various types of sarcoma (myxosarcoma being frequent) have been seen in this region, both pigmented and non-pigmented, and the tumor has been

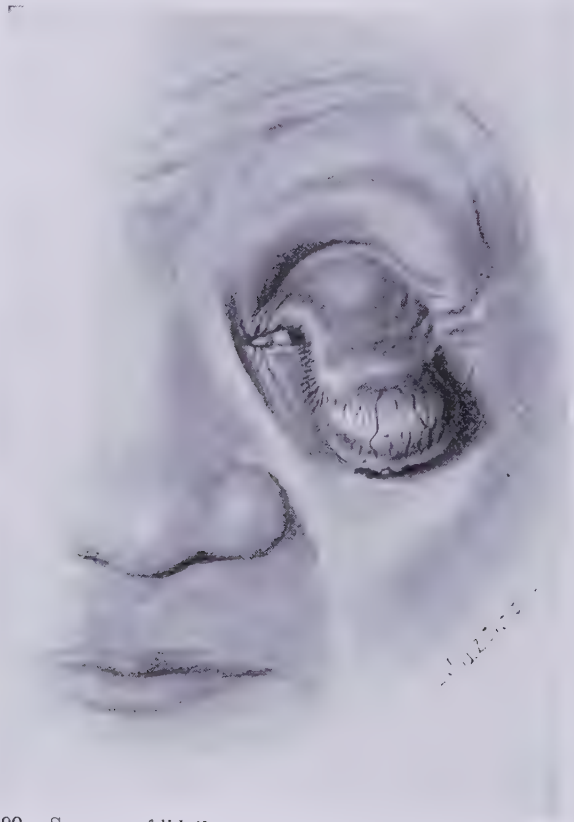


Fig. 90.—Sarcoma of lid (from a patient in the University Hospital).

known to follow a contusion. *Cylindroma*, somewhat allied to sarcoma but less malignant, composed, microscopically, of hyaline cylinders occurs at times in the eyelids of adults, and is of slow growth.

An early removal of the tumor is urgently indicated, but in spite of operation there may be recurrence or metastasis. Radium has been successful in some instances (Callan, Abbe) and after the dissection of the growth from its bed radium treatment is advisable.

Lymphomas occur in the lids and orbits in patients suffering from leukemia. They are often symmetric. They cannot be distinguished

histologically from round-celled sarcomas. Lymphoma of the lower lid may be part of a general lymphomatosis (Coats).

Carcinoma of the eyelid often appears in the form of *rodent ulcer* (Jacob's ulcer), which is a type of epithelial cancer, being, according to F. H. Montgomery, practically a superficial carcinoma of the tubular variety. It is characterized by slow ulceration and non-involvement of the neighboring lymph-glands, and is usually seen in elderly persons.

The growth begins as a pimple, over which a crust appears. Gradually an ulcer forms, which slowly spreads with indurated and elevated edges and, if unchecked, involves all the tissues and destroys the eyeball. Often many years elapse before the ulcer attains any considerable



FIG. 91.—Destruction of eyeball and orbital tissues by a rodent ulcer: five years between the two stages (from a patient in the Philadelphia General Hospital).

size. The most common point of origin is the inner end of the lower lid (Fig. 91).

The slow growth and absence of lymphatic involvement, together with the age of the patient, suffice to distinguish rodent ulcer from a tertiary syphilitic sore.

It may be confounded with lupus, but the latter occurs in younger subjects, is more inflamed and less indurated, the ulcerations proceed from many points, and are generally associated with lupus elsewhere in the body.

Epithelioma with the ordinary clinical characteristics may attack the eyelid, and is one of the commonest tumors of this region. It usually begins at the lid margin, and is more frequent on the lower

than on the upper lid. It not infrequently is situated at the outer commissure and involves both lids (Fig. 92). It may extend to the eyeball and orbital tissues. Microscopically, it consists chiefly of a downgrowth of the interpapillary processes of the rete. The epithelial plugs often contain "cell-nests." According to Ginsberg a certain number of growths recorded as epitheliomas are really *endotheliomas*. *Glandular carcinoma*, having its point of origin either in the Meibomian or in Krause's glands, may also occur in this region.

Treatment.—Certain local remedies, as aristol, chlorate of potassium, injections of pyoktanin, and if the disease is advanced, Canquoin's paste, chloracetic acid, scraping, and the actual cautery were at one time employed to check the ulceration, but they are of no real value. Treatment by *x*-rays and radium associated, if necessary, with surgical measures, is the proper method.

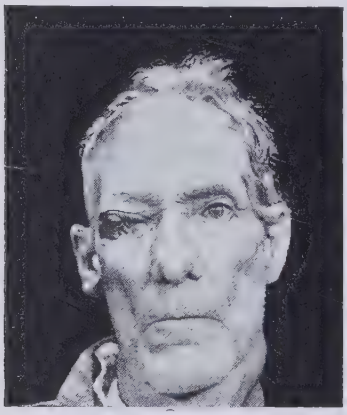


FIG. 92.—Epithelioma of the eyelid (from a patient in the Jefferson Medical College Hospital).

At the author's request Dr. Henry Pancoast, Professor of Roentgenology in the University of Pennsylvania, has prepared the following directions, which will be found useful:

An important consideration in *x*-ray applications in the neighborhood of the orbit is the possible injury to the eye that may result from the exposures. Ulceration and opacity of the cornea, severe conjunctivitis, edema of the conjunctiva and lids, and optic neuritis have been noted. The eye should always be carefully protected.

Epitheliomas involving the lids are frequently troublesome and obstinate, because there is a tendency to allow too little exposure to the rays lest the eye be damaged, or sufficient treatment cannot be given on account of the danger of injuring the ocular coats. If the growth overlies the eyeball, cocain may be introduced, and then a hard-rubber, ivory, or metal eye spatula may be inserted under the lid. All the surrounding healthy parts should be covered by an impenetrable protective. The quality and quantity of rays used are most important. The tube should be soft, such a one which has a resistance equal to 1 or $1\frac{1}{2}$ inches of spark-gap, and it should be placed as near to the area as possible. A current of 1 to 3 milliamperes may be used in the secondary. With the aid of a mechanical spring-interrupter the average duration of each exposure should be from five to ten minutes. On account of the latency of *x*-ray effects many operators advise giving the exposures in series. Four or more applications are made on successive days, and then an interval of several days follows before the next set of exposures is given. With such a technic the necessary number of applications does not entail much danger to the eye. At the same time it should be borne

in mind that preliminary partial or complete excision should be performed when it is not inconsistent with good cosmetic results.

Although, according to Pancoast, excellent results have been obtained in many instances in the treatment of epitheliomas of the eyelids by the use of Roentgen rays, the results from radium he believes are preferable and more certain, and the latter agent can usually be employed to better advantage and with greater safety to the eye. The reaction to radium in the unprotected bulbar conjunctiva is very severe and the eye should, therefore, be adequately protected against the less penetrating *beta rays*. This can readily be accomplished when the application is made near the palpebral margin by inserting a piece of metal such as a Snellen clamp between the lid and the eye after cocainization. Such treatment results in a minimum loss of tissue, and any resulting deformity can be corrected subsequently if necessary. Papillomas may be removed in the same manner. The exact dosage of the



FIG. 93.—Epithelioma of eyelid encroaching on eyeball (from a patient in the Philadelphia General Hospital).

radium depends upon the conditions present. In small superficial epitheliomas of the lid margin, about 25 milligrams of the radium element for half an hour is usually sufficient; the application may be repeated in three weeks if necessary.

In recent years *electric desiccation* has been successfully employed in the treatment of lid carcinomas and also of epibulbar growths and has been highly commended and elaborately practised by W. L. Clark. By "desiccation," according to Burton Chance, who also recommends this procedure, "is meant the dehydration of tissues by means of heat applied in the form of an electric 'flame' produced by a high-frequency current whereby the vitality of the tissue cells is destroyed." The heat is produced by a monopolar electric current of high tension, generated best by a static machine and transformed by suitable appliances. The heat flame, which must not be so intense as to char the tissues, flows from the point of a fine steel needle. "Desiccation" must not be confused with "fulguration," which consists in the indirect destruction of

tissue by the application of a current generated by an induction coil or transformer. The author has recommended the use of electric desiccation in a few cases of lid carcinoma and in one case of epibulbar growth, but prefers *x*-ray and radium treatment, as described in the preceding paragraphs.

Lupus vulgaris is a cellular new growth composed of variously shaped, reddish tubercles, which usually terminate in ulceration and extensive cicatrization. As this disease commonly appears on the face, it may also involve the eyelids.

The process begins in youth, often before puberty, and is slow in its course. The ulcers are apt to start from a number of points which coalesce; their edges are soft, and the discharge is offensive. *Syphilitic ulcers*, on the other hand, are deeper, more excavated, with harder margins, and the course is more rapid. *Tuberculosis of the lid* and *tarsus* is usually secondary to conjunctival tuberculosis; rarely it is primary and may simulate the clinical picture of chalazion (von Hippel).

Treatment.—Local application of caustic paste, erasion with a curet and the actual cautery have been employed, and injections of tuberculin have been recommended.

Lepra.—Leprosy attacks the eyelids very frequently. According to Lopez, two-thirds of those affected with this disease suffer from lesions in this region. These consist of anesthetic patches of color slightly different from that of the surrounding integument, tubercles, loss of the eyelashes and eyebrows, and ectropion and entropion.

Xeroderma Pigmentosum.—According to Greeff, the ocular affections in this disease are found both on the skin of the lids and the mucous membrane of the eye; more rarely on the cornea. The earliest evidences of the disease appear on the face, and particularly on the lids. After certain irritative symptoms an atrophic process develops with areas of pigmentation, and even in the early stage of the disease the cilia fall out and disappear. Later elevations of a warty appearance develop, the epithelial processes of which extend inward and become true carcinomas. Salzmann has described areas of discoloration and atrophy in the uveal tract of the iris. The disease may occur in childhood as well as in adult age, and it is interesting that even in youth carcinoma may develop. L. W. Dean has reported favorable results from the use of cocoa butter locally and injections of autoserum. Thorium and radium have been tried. The carcinomatous tumors may be removed if they are not too numerous.

Elephantiasis arabum, a chronic hypertrophic disease of the skin and subcutaneous tissue, has appeared in the upper eyelid in consequence of an injury, but may also be congenital. According to Cirincione, a distinguishing feature of true elephantiasis of the lids is that at least two lids are involved, and generally four of them. Repeated attacks of erysipelas have etiologic importance. *Elephantiasis telangiectodes*, or that disease which consists in a hypertrophy of the skin and connective tissue, together with fatty tissue and distended vessels, occurs in the upper eyelid as a congenital affection.

Tarsitis, or inflammation of the tarsus, is usually syphilitic in origin, and presents great thickening of the tarsus, owing to diffuse gummatous infiltration (Fig. 94). It may also be due to tuberculosis and to trachoma. As a rule, it is chronic in course; an acute form has been described. The disease may resemble a chronic marginal blepharitis, with the formation of crusts and ulcers at the mouths of the hair-follicles, but differs from the latter condition by the presence of considerable thickening and induration of the tarsus. Alteration of the tarsus, owing to chronic infection of the Meibomian glands, may arise, and has been referred to. Suppurative tarsitis has occurred; the author has recorded one case of this character apparently due to influenza, that is, it developed during an attack of this disease. An ulcerative variety due to syphilis has been described (Morax).

Treatment.—If syphilitic, tarsitis is amenable to the ordinary remedies; if not, much the same treatment described in connection with chronic blepharitis is applicable, especially the use of resolvent ointments.



Fig. 94.—From a photograph of a patient with syphilitic tarsitis under the care of Dr. Randall, in the Children's Hospital.

Blepharospasm, or an involuntary contraction of a portion or the whole of the orbicularis palpebrarum, appears as either a *clonic* or a *tonic* cramp.

The former variety may consist merely in a twitching of a few fibers of the muscle, most commonly in the lower lid, very annoying, and often the cause of undue alarm. It arises from the strain of ametropia and muscle unbalance, prolonged eye use, and deficient amplitude of accommodation. It also occurs in a severe and intractable form and occasions much discomfort and conjunctival irritation, and is associated with spasmodic twitching and spasms of the facial muscles (*facial tic*).

In the *treatment* of the mild types of this affection the prescription of glasses and a general tonic usually suffice.

In stubborn cases fluidextract of gelsemium will occasionally afford relief. Conium internally, and the extract locally, have been recommended. Hypodermic injections of 80 per cent. alcohol at the emergence of the facial nerve have been tried and satisfactory results have been reported. It is advisable to explore the nasopharynx. In one remarkable case under the author's care the correction of a refractive error associated with straightening a deviated septum resulted in practically entire relief, and in another treatment of a hypertrophied turbinate bone was equally successful. The teeth should be carefully examined. A lotion of 1 per cent. holocain in physiologic salt solution is useful.

Children are often affected, especially during their early school years, with undue winking of the eyelids, associated at times with jerky

movements of the facial and other muscles. This form of nervous disorder was designated by Weir Mitchell *habit chorea*.[1] Almost invariably, blepharitis, follicular and phlyctenular conjunctivitis, and errors of refraction and heterophoria are exciting causes. Long-continued blepharospasm, especially in children with phlyctenular conjunctivitis, may give rise to *lid edema*, due to pressure of the contracted orbicularis on the palpebral veins. Suitable glasses and appropriate local remedies, together with the exhibition of Fowler's solution, are indicated.

Tonic cramp of the orbicularis follows the introduction of foreign bodies into the eye, the presence of inflammations of the cornea and conjunctiva, and fissures at the angles of the lids, and depends upon irritation of the peripheral trigeminal filaments.

More rarely a persistent lid cramp occurs, without obvious cause, and is unrelieved for weeks and even months. When the eyes are finally opened, there may be temporary blindness, without corresponding ophthalmoscopic changes; or permanent loss of vision, with gross lesions in the eye-ground.

Blepharospasm, both clonic and tonic, usually the latter, is not an uncommon manifestation in the subjects of hysteria (see also pp. 582, 583) as is also *spastic ptosis*, which depends upon a cramp or spasm of the palpebral portion of the orbicularis. Tonic blepharospasm was frequently noted among the so-called "shell-shock" cases during the war.

The *treatment* demands the removal of any peripherally exciting cause –fissure, foreign bodies, phlyctenules, etc. Hypodermic injections of morphin have been used to control the trigeminal irritation, and in severe cases section of the supra-orbital nerve has been performed. Conium and gelsemium in the form of the fluidextract may be tried. They should be pushed to the point of tolerance. The hypodermic use of alcohol has been referred to. Hysterical blepharospasm is curable by "suggestion" and the general measures suited to this psychosis.

Paralysis of the orbicularis, chiefly noticeable when the patient endeavors to close the lids, which are then only partly approximated, is due to an affection of the facial nerve. Epiphora is apt to be marked because of the sagging of the inner half of the lid (*paralytic ectropion*). There is always danger of *exposure keratitis* (see also p. 293). Facial palsy and hence paralysis of the orbicularis of interosseous or external origin (Bell's palsy) may be due to exposure to cold, to aural diseases, especially suppuration of the middle ear, to injury, to fracture of the skull, and to operation on the parotid and middle ear. The author has observed bilateral paralysis of the orbicularis following fracture of the skull with only slight involvement of the lower facial area. In some cases a toxic neuritis or perineuritis caused, for instance, by syphilis or acute specific infectious processes is responsible for paralysis of the orbicularis. More rarely the lesion has a central situation.

Treatment.—After removal of the cause this is largely symptomatic and may include electricity. The eye should be covered to avoid exposure keratitis.

[1] Gowers gave the name "habit spasm" to this affection.

Ptosis (*blepharoptosis*) is that condition in which the upper lid droops entirely or partially over the eyeball, and cannot be voluntarily raised. It is either congenital (see page 173) or acquired by reason of the development of fatty or other accumulations in the connective tissue of the lid (*pseudoptosis*, see page 185), or it arises from paralysis of the oculomotor nerve, and in rare instances from lesion of its cortical center (*paralytic ptosis*). Slight ptosis may follow paralysis of the sympathetic nerve, because this supplies the superior tarsal muscle of Müller (see also page 411). Ptosis also occurs as the result of injury of the levator.

In some cases of unilateral congenital ptosis, usually on the left side, while the eyelid cannot be voluntarily raised, it is elevated when

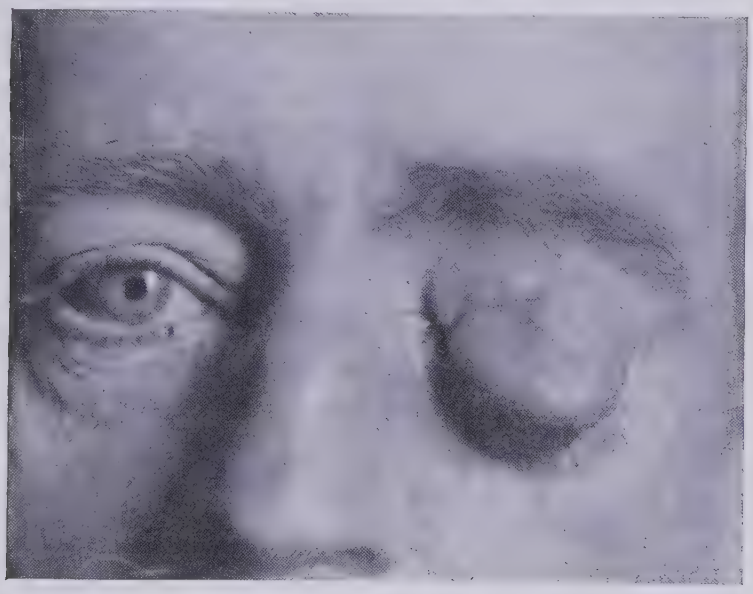


Fig. 95.—Ptosis with edema of tissues, the result of laceration of the lid and insertion of the levator.

the jaw is moved during eating (contraction of the levator in association with the external pterygoid or *jaw-winking*) (see also Ocular Palsies).

It is convenient to make reference in this place to other associations in muscle action. Thus, there may be contraction of the orbicularis with movements of the jaw, contraction of the levator with abduction and adduction, and, as reported by Zentmayer, contraction of the frontalis with abduction.

Treatment.—The medicinal treatment of ptosis consists in the exhibition of those remedies which control the supposed cause of the palsy—mercury and iodids in syphilis, salicylic acid in rheumatism.

The surgical treatment will be found on page 691.

Blepharochalasis, or relaxation of the skin of the lid, due to atrophy of the intercellular tissue, has been described by Fuchs and other writers.

The skin of the lid is thin, much wrinkled, and its superficial veins are dilated. The condition may be remedied by excising appropriate portions of the relaxed tissue and uniting the cut edges with sutures. Verhoeff and Jonas Friedenwald have demonstrated that there is a diseased condition of the endothelium of the capillaries and veins manifesting itself in swelling and proliferation of endothelial cells.

Lagophthalmos, or an inability to close the eyelids completely (total lagophthalmos is rare), is either paralytic or non-paralytic, and usually results from paralysis of the facial nerve, as already described (page 194), but also occurs as the result of loss of lid tissue, in ectropion, in tumors of the orbit, exophthalmic goiter, staphyloma, and as a congenital defect (see page 171). Failure of the lids to come in contact with the globe at the outer canthus has been recorded as a congenital condition. Widening of the palpebral fissure and drawing up of the upper lid due to *spasm* of the *superior tarsal muscle* of Müller, such, for instance, as is produced by the instillation of cocain, must not be mistaken for lagophthalmos.

The chief danger of the affection is ulceration of the cornea from exposure, rendered all the more certain should disease of the trigeminus also exist.

Treatment.—In paralytic lagophthalmos the primary cause of the affection must be treated: in the non-paralytic varieties, and in any form in which the vitality of the cornea is threatened by its exposure, the operation of tarsorrhaphy may be employed (see page 696).

Symblepharon,[1] or a cohesion between the eyelid and the ball, may be complete or partial, acquired or congenital (see page 171). The most usual causes are injuries, especially burns with acids, lime, or molten metal (see page 264). Symblepharon also follows diphtheritic conjunctivitis, trachoma, pemphigus, and occasionally purulent conjunctivitis; but the shortening of the conjunctival sulcus, which is due to a species of drying of the conjunctiva, presently to be described, must not be confounded with a true symblepharon.

The attachment may consist of comparatively slight bands between the conjunctival surface of the lid and ball, or one or both eyelids may be entirely adherent to the globe. This is particularly the case after burns (molten metal, lime, and acid), and as the result of pemphigus, the cornea may also be involved in the cicatrical union, and vision be seriously disturbed. The lower lid is most usually involved in the process; the upper may also participate (Fig. 96).

Ankyloblepharon, or that condition in which the borders of the two lids have grown together, may be congenital or acquired, and, like the preceding affection, partial or complete.

The same causes which originate symblepharon are active, and varieties have been described in which the union takes place not by a growing together of the lids, but by the organization of a membrane, the result of croupous conjunctivitis.

[1] Symblepharon really belongs to diseases of the conjunctiva, but its description is conveniently inserted in this place.

Blepharophimosis is that condition which develops from a contraction of the outer commissure of the lids, and results in shortening of the palpebral fissure.

It is commonly seen in cases of long-standing conjunctivitis with irritating secretions; for instance, in chronic conjunctivitis and in some of the forms of trachoma.

Treatment.—After an injury, or during the course of a local disease, likely to result in one of these complications, scrupulous care must be exercised to avoid it. The formation of granulation tissue may be broken up with a probe, and it has been advised to place a piece of gold-beater's skin or the thin skin from the inner surface of an egg-shell (Coover) between the lid and the ball to prevent adhesions. It is very difficult to keep an artificial protecting membrane between the raw areas of bulbar and palpebral conjunctiva and the daily passage of a blunt probe between the surfaces which are tending to adhere is more

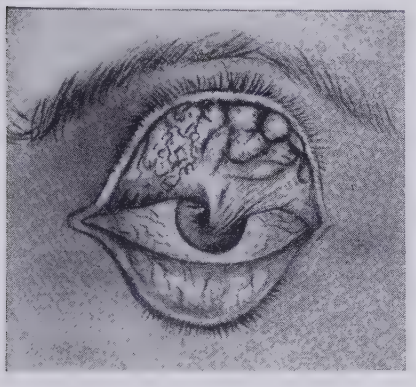


FIG. 96.—Symblepharon, the sequel of purulent conjunctivitis (from a patient in the Philadelphia General Hospital).



FIG. 97.—Symblepharon, the result of a lime burn (from a patient in the University Hospital).

effective than any of the other procedures. Boric acid salve or bichlorid ointment (White's ointment, p. 762) should be placed on the conjunctival culdesac.

The surgical treatment of these affections is described on page 717.

Trichiasis; Distichiasis.—*Trichiasis* is that affection in which the lashes are misplaced and turn inward against the eyeball; *distichiasis* is that condition in which incurved rows of supplementary cilia are developed from the intermarginal part, close to the opening of the tarsal glands.

The most usual causes of trichiasis are chronic inflammations of the lid borders and conjunctiva—blepharitis and trachoma.[1] Distichiasis, in rare instances, is congenital, or develops about the age of puberty.

[1] Raehlmann taught that trichiasis hairs, or "false cilia," are developed from the epithelial covering of the lid margin in consequence of marginal blepharitis the result of granular conjunctivitis.

The cilia rubbing against the cornea produce constant irritation and may cause ulceration.

Treatment.—If not too numerous, the lashes having a faulty direction should be removed with cilium forceps, and when they grow again, the procedure repeated; their reappearance may sometimes be prevented by destruction of the hair-follicles by galvanopuncture. Other operations consist of strangulation of the roots of the incurved lashes by a subcutaneous ligature, excision, and the various modifications of a single and double transplantation of the entire ciliary border (see chapter on Operations).

Alopecia of the eyelids, the loss of the lashes depending upon the fact that the patient, usually a hysteric girl, systematically pulls out the cilia, has been described by H. Gifford. The author has seen several cases of this character.

Sudden *turning gray of the eyelashes* has been recorded by Hirschberg after phlyctenular disease, and has occurred in sympathetic oph-

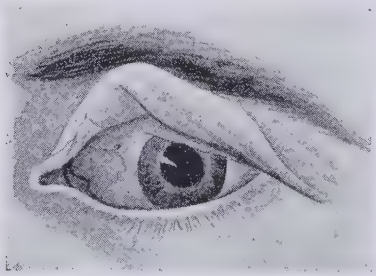


Fig. 98.—Ectropion of the upper lid, the result of an injury to the brow and subsequent caries of the margin of the orbit (from a patient in the Philadelphia General Hospital).

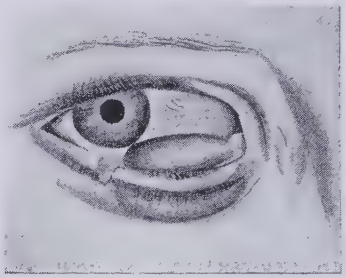


Fig. 99.—Ectropion of the lower lid, the result of a wound from the tine of a fork; marked chronic conjunctival edema (from a patient in the Children's Hospital).

thalmia and iridocyclitis. *Premature grayness* of the cilia, sometimes temporary, has also been reported. Frequent exposure to *x*-rays may cause the eyelashes and eyebrows to turn gray and to fall out.

Entropion, or inversion of the lid, like trichiasis, is most commonly caused in an *organic* form by trachoma, and also follows essential shrinking of the conjunctiva and diphtheritic conjunctivitis. Entropion and trichiasis are often associated.

Two other varieties of entropion are described—*muscular* and *bulbar*. The former is sometimes present at birth from undue development of the orbicularis, and also occurs in a spasmodic type, under the influence of conjunctivitis, keratitis, foreign bodies and sometimes after operations and bandaging of the lids; the latter is a falling in of the lids when the eyeball is shrunken or absent.

Treatment.—The spasmodic varieties will usually subside if the exciting cause can be removed. In temporary entropion the lid may be painted with flexible collodion, which, by its contraction, draws out

the inverted border, or, having everted the lid, it may be held in place with a longitudinal strip of plaster which is fastened to the cheek. Often a spasmodic entropion, especially in elderly people, must be remedied by an operation (page 700). The organic varieties of the disorder require one or other of the operations described on page 699.

Ectropion, or eversion of the lid with exposure of the conjunctival surface, is either partial or complete. The disorder is divided into the *acute* or *muscular* and the *chronic* form, or that which results from organic changes.

Acute ectropion usually occurs in children with conjunctivitis and in diseases of the cornea with blepharospasm, when the lids, during examination, become everted and remain so until replaced. One form of partial muscular ectropion is produced by facial palsy.



FIG. 100.—Ectropion of the lower lid, caused by caries of the malar bone (from a patient in the Philadelphia General Hospital).



FIG. 101.—Ectropion of the upper lid, which is completely everted, from syphilitic periostitis of the orbit (from a patient in the Philadelphia General Hospital).

The common causes of the second, or chronic, form of ectropion are wounds and lesions, for example, such as are caused by dog-bites, by laceration of the lid by a sharp instrument, by shrapnel injuries, by burns and subsequent cicatricial contraction, by chronic inflammatory conditions of the ciliary margin, by ulceration of the lids as in lupus, and by caries of the orbital border and malar bone. The lower lid is more frequently involved than the upper, but ectropion is also seen in the latter position.

Treatment. —This varies with the type and degree of the ectropion. In the spasmodic forms simple replacement of the everted lids suffices; in slightly marked grades, with some eversion of the lacrimal punctum, the canaliculus should be partly slit and, if necessary, the nasal duct should be probed; sometimes the condition is favorably influenced by

painting the everted conjunctiva, which usually is thickened and roughened, with an astringent; for example, tannin and glycerin or



FIG. 102.—Ectropion of the lower lid following lupus. The scar on the cheek is faintly seen (from a patient in the University Hospital).



FIG. 103.—Inferior ectropion from contraction of tissue after a wound over malar bone (from a patient in U. S. General Hospital No. 11).

boroglycerid. The organic types of the disorder require a plastic operation for the relief of the deformity (see chapter on Operations).

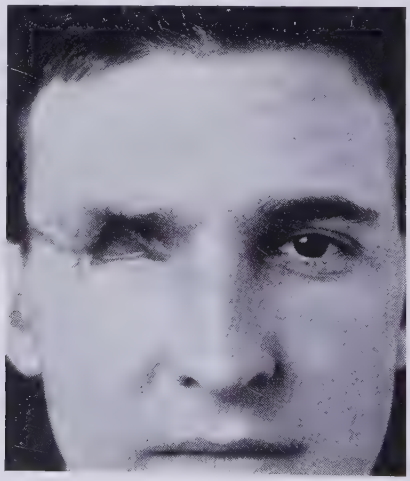


FIG. 104.—Superior ectropion from shrapnel wound of lid and brow—anophthalmos (from a patient in U. S. General Hospital No. 11).

Certain diseases of the eyelids depend upon disorders of the sebaceous glands and sweat-glands.

Seborrhea, or that disorder of the sebaceous glands during which their secretion is altered and forms an oily coating on the skin, sometimes coexisting with crusts and epithelial scales, is also seen upon the eyelids. It is usually associated with a similar process in the scalp and eyebrow, and when specially localized upon the ciliary margins creates one of the forms of blepharitis already described.

Treatment.—Proper hygiene, cod-liver oil, iron and arsenic, removal of the accumulated sebum by frequent washings, and the application of sulphur and mercurial ointments comprise the most efficient methods of treatment.

Milium.—Milia, or small yellowish elevations, consisting of an accumulation of sebum within the distended but closed sebaceous glands, are common upon the eyelids. They often develop about the age of puberty.

They are caused by improper care of the skin, and may be connected with general constitutional disturbances, dyspepsia, and constipation. They should be opened with a knife or needle and the contents evacuated.

Molluscum contagiosum (**molluscum sebaceum**) is a disease of the sebaceous glands (according to some authors, of the rete mucosum) characterized by the appearance of rounded papules, about the size of a pea, and of a waxy color. The eyelids are a favorite situation. It may be the cause of chronic conjunctivitis (page 249).

The disorder occurs chiefly among ill-nourished children, is believed by many to be contagious, and may arise as an epidemic in homes and asylums. According to some observers, the affection is caused by a parasite belonging to the class *coccidia*, and really is a form of *contagious epithelioma*. Muetze's investigations indicate that the "molluscum corpuscles" are the result of a degeneration of the epithelial cells caused by the contagion, the nature of which is uncertain.

Treatment.—Each molluscum should be incised and its contents forced out.

Ephidrosis (*hyperidrosis*), or an increased flow of sweat, has in rare instances been observed as a local disorder of the sweat-glands of the eyelids. In cases of unilateral sweating of the face the lids necessarily participate. *Retention cysts* of the sweat-glands, in the form of round, sharply circumscribed elevations, have been observed by von Michel.

Chromidrosis (*seborrhœa nigricans*), or the formation of a variously colored secretion from functionally disordered sweat-glands, is sometimes located upon the eyelids. It then receives the name of *palpebral chromidrosis*, and consists of a bluish-black discoloration, usually upon the lower lid, which is somewhat oleaginous and can be wiped away.

It is probably genuine in rare instances; in others it is believed to represent an act of deception practised by an hysteric subject or to be due to a deposit of dust upon the surface of the skin affected with seborrhea. Young women are usually those affected.

The *treatment* should consist in general invigorating methods calculated to remove anemia, debility, or nervous disturbances. Locally, lead-water and glycerin are recommended.

Sebaceous cysts occur in the eyelids, most frequently in the outer part, and also in the eyebrow. In the latter situation they sometimes are deeply seated, tightly adherent to the periosteum, and may extend some distance into the orbit. *Dermoid cysts* are also found in this region. Their removal by an ordinary dissection is usually unattended with difficulty.

Injuries of the Eyelids.—Incised, lacerated, punctured, and contused wounds, edema, emphysema, and ecchymosis of the lids are the ordinary results of accidents and injuries.

Wounds.—The type of a wound depends largely upon the character of the implement which has inflicted it, and may vary from a simple and superficial incision to a deep cut which penetrates the tissues



Fig. 105.—Dermoid cyst of the eyebrow.

of the lid and injures the structures of the eyeball. In like manner a laceration may be small and unimportant, or may be so extensive as to tear the eyelid from its attachments. Incised wounds in the line of the direction of the fibers of the orbicularis result in the least visible scar, owing to the absence of gaping. Such injuries are often inflicted by broken glass (a broken spectacle lens, for example), or china, by a knife or other sharp instrument, and by a thin sliver of metal. During the past war eyelid wounds, often in association with grave facial wounds, were extremely common.

Treatment.—Approximation of the edges of the wound should be secured with catgut, horsehair, or fine silk sutures. Even considerable laceration may heal with very little deformity if the proper technic is adopted. The important points are that the sutures should be applied as early as possible after the injury, that if the wound is a penetrating one the conjunctiva should be carefully sutured and the line of lashes restored before the skin wound is approximated, that, especially neces-

sary in the case of multiple wounds, all tissue should be retained the retention of which is feasible, and that the stitches should be neatly inserted and with due regard to a coaptation of the lacerated parts in their proper positions.

Edema usually occurs as the sequel of a blow, owing to the loose connective tissue of the eyelids, which is readily distended (see also page 173).

Treatment.—The application of evaporating lotions, for example, dilute lead-water and laudanum, associated, if the swelling is great, with a pressure bandage is a measure which will afford relief.

Emphysema of the lids is observed when a fracture of the orbit permits air to escape into the cellular tissue through a communication thus produced with the ethmoidal or frontal sinus. A soft swelling, crackling to the touch, is the result, which increases in degree when the patient blows his nose and forces the air through the fissured bone. The eyelids may participate in the emphysema of the neck and face sometimes seen after tracheotomy or after stab-wounds of the chest.

Ecchymosis of the lids, or a collection of blood in the connective tissue, in its simplest variety constitutes the familiar "black eye," the common result of a blow. A gradual absorption of the effused blood takes place, requiring a week or longer for its completion, but the skin may retain its black-and-blue stain for a greater period of time.

Ecchymosis may be due to fracture of the base of the skull, and may be associated with emphysema if a fracture has involved the frontal or ethmoidal cells.

Treatment.—Emphysema will gradually subside without local treatment; if the swelling is severe, it has been recommended to prick the tissues and allow the air to escape.

Ecchymosis should be treated by frequent applications of cold water, lead-water and laudanum, or diluted white extract of hamamelis. If discoloration remains for a long time, the "eye may be painted." The practice of applying leeches or incising the swollen lid and sucking out the contained blood is pernicious.

Foreign Bodies in the Eyelids.—Fragments of glass, shot, pieces of iron or steel, portions of wire, particles of stone, and splinters of wood may penetrate the tissue of the eyelids and occasionally remain undetected for long periods of time. They may become encysted or, as the result of infection, give rise to a *lid abscess*. If the foreign body is composed of iron or steel it may be removed by means of a magnet.

Burns of the eyelids are commonly caused by hot water, caustics (lye and lime), acids, and by the explosion of powder.

The first agent produces the ordinary vesication, and the treatment should consist in the application of oil, while the pain may be materially relieved by using locally a lotion of carbonate of soda or, better, the moistened powder itself.

Burns caused by the other materials are especially dangerous on account of the almost invariable involvement of the cornea and conjunctiva (see page 264). Immediately after a powder burn all loose

powder should be removed. Deeply embedded grains can sometimes be picked out with a fine needle; usually a spot of discoloration remains. E. Jackson has suggested that large powder grains may be destroyed by touching them with a fine electrocautery needle, to be followed by the ordinary applications suited to burns. Peroxid of hydrogen is a most efficient remedy for powder burns; the affected skin areas should be vigorously rubbed with this medicament in full strength or in a solution of 3 parts to 1 part of glycerin.

CHAPTER VI

DISEASES OF THE CONJUNCTIVA

Congenital Anomalies of the Conjunctiva.—In addition to dermoid tumors (see page 317) certain thickenings of the conjunctiva of congenital origin have been reported. The latter resemble pterygia and extend between the fissures of the lid (Strawbridge). If necessary, excision may be performed (see also Epitarsus, page 173).

Hyperemia of the conjunctiva (*dry catarrh; hyperæmia palpebraris*) is characterized by an injection of the vessels, chiefly of the palpebral conjunctiva, but rarely affecting the ocular expansion of the membrane. The posterior conjunctival vessels (System I) are involved, but not to the same extent that they are in conjunctivitis. Both an acute and a chronic form exist.

Causes.—The strain of uncorrected ametropia furnishes a large contingent of these cases. Beginning presbyopia, especially in those persons who are disinclined to use glasses, and hyperemia of the conjunctiva are often associated; it also occurs in incipient cataract and slight opacities of the cornea, as the result of the effort to obtain clear images.

Local irritants, as dust, foreign bodies, misplaced cilia, calcareous concretions, tobacco smoke, cold winds, etc., are common causes, and the abuse of alcohol originates many cases. The condition may also arise in the eyes of those much exposed to bright light, to great heat—for example, in iron foundries and among workers in *x*-ray rooms. Patients with prominent eyeballs are more liable to hyperemia than those whose eyes are more deeply placed.

Nasal catarrh, lacrimal obstruction, and marginal blepharitis are frequently accompanied by chronic hyperemia of the conjunctiva, which is much aggravated by the establishment of an acute coryza or "hay-fever."

Finally, certain acute hyperemias, which may be recurrent, appear in the form of vasomotor disturbances, and arise under the influence of metabolic disorders, especially gout. Hyperemia of the conjunctiva also occasionally occurs in anemia and chlorosis in place of a pallid membrane, and may be associated with trigeminal neuralgia and migraine. Chronic conjunctival congestion has been attributed to gastro-intestinal auto-intoxication (J. F. Shoemaker), and has been observed in association with protein sensitization and the ingestion of certain foods.

Symptoms.—Direct inspection reveals the congestion of the vessels, not sufficient to produce the velvety appearance seen in conjunctivitis and unaccompanied by any discharge. Swelling of the conjunctival follicles may be present, especially if the hyperemia is of long standing.

There are photophobia, some lacrimation, a hot, stinging sensation aggravated by the use of the eyes, which readily "water" and grow uncomfortable, especially in artificial light.

Treatment.—This requires the correction of refractive error. Removal of exciting local causes and attention to the anterior and posterior nares are necessary. Patency of the canaliculi and of the lacrimal passages should be secured.

Locally, boric acid, gr. x to f℥j (0.65 gm. to 30 c.c.), or biborate of soda, gr. v (0.324 gm.), camphor water, f℥j (30 c.c.), and distilled water, f℥j (30 c.c.), may be applied. More active astringents, as alum, tannin, and zinc, are sometimes employed, and stimulating drops, for instance boric acid solution, to which a few drops of alcohol have been added, are useful. Nitrate of silver is not advisable; argyrol (10 per cent.) is sometimes helpful. Douching the eyes with hot or cold water is a valuable adjuvant. Temporary blanching of the conjunctiva for the purpose of differentiating deep and superficial injection may be secured with adrenalin (1 : 10,000) and preparations of suprarenal extract, but they are not advisable as frequent or constant applications. If there is reason to suspect any general trouble, for example, gout, this must receive attention and the diet list must be corrected.

Conjunctivitis.—The conjunctiva is liable to various grades and types of *inflammation* which have certain symptoms in common: (1) Photophobia, not constantly present in all varieties, but commonly seen at some time during the course of the disease; (2) increased and usually altered secretion; (3) a changed appearance in the membrane, varying from a general injection of the blood-vessels and slight velvety opacity to the development of special pathologic products or the formation of false membrane.

The generic term *conjunctivitis* (*ophthalmia* of the older writers) is applicable to this entire group of diseases. Although bacteriologic examinations have given rise to a classification of conjunctivitis which has been recommended in place of the older arrangement, our knowledge is not yet sufficiently exact to make it expedient to banish entirely descriptions based upon clinical appearances. It should be remembered that the normal conjunctiva always contains bacteria, a number of varieties having been isolated. Comparatively few of them should be classified as at all pathogenic (Weeks); but non-pathogenic bacteria may become harmful if the tissues in which they exist are bruised or irritated. According to Axenfeld, the *xerosis bacillus* and non-virulent, or only slightly virulent, *Staphylococcus albus* are practically always present in the normal conjunctiva; other organisms occasionally found are *Staphylococcus pyogenes aureus* and *albus*, *pneumococcus*, *Streptococcus pyogenes* (rare), *diplobacillus* and *influenza bacillus* (uncommon), *Bacillus subtilis*, and *sarcinæ*.[1]

[1] MacCallan has classified the various forms of conjunctivitis encountered in Egypt from the bacteriologic standpoint into four groups: the gonococcal group, the Koch-Weeks group, the Morax-Axenfeld group, and the group due to other organisms.

Simple Conjunctivitis (*Catarrhal Conjunctivitis or Ophthalmia*).—This is an inflammatory disease of the conjunctiva, characterized by congestion, loss in the transparency of the palpebral conjunctiva, some dread of light and spasm of the lids, and a discharge sufficient only to glue the lids in the morning, or freer and mucopurulent.

Causes.—The etiology is made evident by observing certain varieties:

Associated conjunctivitis is seen with eczema, facial erysipelas, impetigo contagiosa, nasal catarrh, bronchitis, and rheumatism and typhoid fever (typhoid bacilli have been found in the secretion of conjunctivitis in this disease). In *acne rosacea conjunctivitis* minute nodules form at the limbus; moderate irritation supervenes; subsidence takes place, but recurrence is frequent. The condition resembles somewhat phlyctenular conjunctivitis. It arises in adults who are the subject of acne rosacea. *Exanthematous conjunctivitis*, which accompanies or follows measles, scarlet fever, and smallpox, may be included in this list. In the conjunctivitis of epidemic meningitis McKee has found the *meningococcus*. This conjunctivitis is an early symptom, and McKee thinks the bacteria may be found in the conjunctival secretion before they can be recovered from the cerebrospinal fluid. Conjunctivitis, associated with excessive lacrimation, has been observed after thyroidectomy and in cases of myxedema (T. F. Shoemaker).

Mechanical conjunctivitis results from exposure to wind, dust, and traumatism (*toxic conjunctivitis*, see page 250).

Symptomatic conjunctivitis may arise from the strain of ametropia, and is analogous to ordinary hyperemia.

Micro-organisms (staphylococci, streptococci, pneumococci) are present in severe types and explain the contagion; neglected hyperemias and the presence of follicular granulations increase the susceptibility to infection, and scrofulous subjects are peculiarly liable to the disease. Occasionally a stubborn conjunctivitis is encountered from the secretion of which pure cultures of staphylococci may be obtained, and which, therefore, has been called *Staphylococcus conjunctivitis*. McKee has described a variety of mucopurulent conjunctivitis due to a new pathogenic organism which somewhat resembles the influenza bacillus. One variety of conjunctivitis is due to the *Micrococcus catarrhalis*, and is often accompanied by rhinopharyngitis. It may be comparatively mild in its manifestations, but severe and decidedly mucopurulent and sometimes epidemic types occur. In this connection it is important to remember that the Micrococcus catarrhalis resembles the gonococcus in morphology and staining. Conjunctivitis may also be caused by the presence of the *meningococcus*, *Bacterium coli* (Axenfeld), *ozena bacillus*, and *Bacillus subtilis* (Gourfein).

Symptoms.—The secretion is at first watery and, by running over the edge of the lids, may excoriate the surrounding skin, which shows injection of its superficial veins. In certain individuals the lids, especially along their palpebral margins, are slightly edematous.

The secretion soon becomes mucous or mucopurulent and, accord-

ing to the grade of the inflammation, gathers in a slightly frothy material only at the commissural angles, or is more freely secreted.

There are a general hyperemia and loss in the transparency of the tarsal conjunctiva, in which the posterior conjunctival vessels (System I) are concerned, and later of the fornix, caruncle, and semilunar folds.

Although vision is not usually affected, some secretion may be adherent to the cornea and produce the same haziness of sight that would be present on looking through a dirty glass; and artificial lights, which are most uncomfortable at all times, appear fringed with colored borders.

Photophobia may be entirely absent, or exist in marked degree in those varieties which complicate measles, or which are associated with the development of superficial *ulcers on the cornea.* All ages of life are liable to catarrhal conjunctivitis, but the majority of the cases are seen in children and young people.

Prognosis and Duration.—The prognosis of ordinary catarrhal conjunctivitis is perfectly good, and the process usually subsides in a few days. One or both eyes may be affected. In other types the duration may be prolonged and the manifestations severe, sometimes because of neglect and improper medication (see also page 212).

Acute Contagious (Communicable) Conjunctivitis (*Acute Mucopurulent Conjunctivitis; Epidemic Conjunctival Catarrh; "Pink-eye"; Koch-Weeks' Bacillus Conjunctivitis*).—This form of conjunctivitis may be classified as the severe and epidemic type of the variety of conjunctival affection just described. By some writers it is considered as a distinct disease.

Etiology.—The majority of cases are caused by a small bacillus discovered independently by Koch in the acute conjunctivitis of Egypt, and by Dr. John E. Weeks in New York, and studied by Morax and others in Europe. This bacillus resembles that of mouse-septicemia, and measures 1 to 2 μ in length and about 0.25 μ in breadth. It is often associated with a clubbed bacillus (xerosis bacillus). It is stained readily by ordinary anilin dyes. Some observers have maintained, but have not demonstrated, that the Koch-Weeks' bacillus and the influenza bacillus are identical, and that acute contagious conjunctivitis is a manifestation of influenza. The disease may occur at any age except, perhaps, during the first few days of life, and is wide-spread over the world. It is commonest in warm and changeable weather (the fall and spring), is markedly communicable, and passes rapidly from one member of a household to another.

Symptoms.—The period of incubation is about thirty-six hours, and the disease begins with the symptoms of a mild catarrhal conjunctivitis, but usually on the third day develops into a severe form of conjunctivitis, in which the entire conjunctiva is deeply injected and small hemorrhages may be observed (*hemorrhagic catarrhal conjunctivitis*), the swelling of the conjunctival membrane being noticeable in opaque velvety layers, especially in the region of the retrotarsal fold. Sometimes the bulbar conjunctiva is chemotic, sometimes brightly

injected. The lids are glued together in the morning, and occasionally they are decidedly swollen and edematous; the eyes are hot and heavy, and feel as though they contained sand. The secretion is at first thick and ropy, and may be gathered into long strings of mucopus. Later, in some cases, the discharge becomes distinctly purulent. The acute stage lasts from four to ten days, and recovery may be expected in about two weeks. Toward the end of the disease, or in what may be known as the subacute stage, the retrotarsal folds are swollen and the papillary body is enlarged. Follicular hypertrophy is at times also observed, and if care is not taken the affection may last for a long time. Both eyes are almost always involved, sometimes simultaneously and sometimes one a day or two in advance of its fellow. Corneal complications, that is, ulcers occasionally occur (Morax, Shumway). Hypopyon has been observed (Morax).

Diagnosis and Prognosis.—The actual diagnosis depends upon microscopic examination and the finding of the specific micro-organism, but the clinical signs are very striking, particularly the character of the secretion, with its tendency to gather in yellowish masses toward the inner canthus. If the disease is known to be epidemic at the time, or if it has passed from one member of the family to another, the diagnosis becomes still more certain.

The prognosis is good in the majority of the cases, although relapses and recurrences are common, and one attack does not create immunity. The affection through neglect, however, may prove exceedingly troublesome, and tends to attack all members of a household, a fact which, in asylums and similar institutions, may be of serious import.

Pneumococcus Conjunctivitis.—This form of conjunctivitis, due to the Fränkel-Weichselbaum diplococcus (pneumococcus), was originally described by Parinaud and Morax, and was supposed by them to be an affection of early childhood; indeed, it is more common in children than in adults, but no age of life is exempt. While it is not as communicable as the Koch-Weeks' bacillus conjunctivitis, its communicable nature is well established, and it may be transferred from one eye to another, from one person to a second, and as Gasparrini, Harold Gifford, Veasey, the author, and a number of other observers have noted, may appear in epidemic form, although usually not so extensively or frequently as the Koch-Weeks' conjunctivitis. However, very marked epidemics of this type of conjunctivitis have been observed, some for example in camps and cantonments during the late war. Bacterial findings vary according to time and place. Thus, in Philadelphia during the prevalence of acute conjunctivitis the pneumococcus usually is more in evidence than the Koch-Weeks' bacillus, while the latter micro-organism is common in New York. According to Axenfeld, pneumococcus conjunctivitis is more frequent in northern countries and in cold months of the year than other forms of mucopurulent conjunctivitis. Very rarely it is associated with pneumonia; a coryza may accompany or precede it.

Symptoms.—Usually it begins as an ordinary conjunctivitis, the

conjunctiva being reddened and secreting a rather thin mucopurulent discharge, in which small flocculent masses float. The upper lid is edematous and pinkish in color, the lashes lightly matted, and later the discharge becomes thicker and more purulent, and sometimes resembles, in severe cases, that found in purulent conjunctivitis. Small subconjunctival hemorrhages may appear. The clinical manifestations of this form of conjunctivitis are often difficult to distinguish from Koch-Weeks' bacillus conjunctivitis. According to Gasparrini, in pneumococcus conjunctivitis a fine pellicle of fibrin can be wiped from the everted upper tarsus, which is not met with in Weeks' bacillus conjunctivitis. Generally the disease lasts from six to ten days, and the prognosis is favorable; rarely corneal complications in the form of ulcers have been noted; occasionally iritis develops, due to absorption of the toxin, without implication of the cornea (Axenfeld).

Influenza Bacillus Conjunctivitis.—This form of conjunctivitis, due to the influenza bacillus (Pfeiffer's bacillus), has been especially studied by Zur Nedden, Morax, Jundell, and, in this country, by Arnold Knapp. Clinically, the manifestations of the disease are not severe: it is characterized by a copious, thin discharge, and affects chiefly the conjunctiva of the lids and retrotarsal folds. The majority of cases have occurred in young children and infants, in whom it is more severe than in adults, who are rarely attacked. Although the local disease presents a favorable prognosis, and usually disappears in from ten to fourteen days, it may be associated with rhinotracheitis, dacryocystitis, and inflammation of the middle ear. Arnold Knapp has described a *pseudomembranous form* of influenza bacillus conjunctivitis of great severity, which may cause perforation of the cornea.

Swimming-bath Conjunctivitis.—This form of conjunctivitis is encountered among those who frequent public baths. In many instances the symptoms are analogous to those which occur in catarrhal conjunctivitis, although the discharge is less abundant. Often the bulbar conjunctiva is coarsely injected. In two local epidemics of this disease in two large schools equipped with swimming-pools investigated by the author the micro-organism found was in one instance the pneumococcus, and in another an unidentified bacillus. Sometimes the affection resembles acute trachoma, and Paderstein has been able to demonstrate "inclusion bodies."

Diplobacillus Conjunctivitis (*Morax-Axenfeld Bacillus Conjunctivitis, Angular Conjunctivitis, Subacute Conjunctivitis*).—This form of conjunctivitis, due to a diplobacillus 2 to 3 μ in breadth, often occurring in chains, was originally described by Morax and by Axenfeld abroad, and later was studied by Harold Gifford in this country.

Symptoms.—One variety of the disease is insidious in character, and runs a rather tedious course, during which the main symptoms are redness and slight induration of the edges of the lids, particularly of the commissural angles, and congestion of the neighboring conjunctiva. In other words, the signs are those of a *blepharoconjunctivitis* or *angular conjunctivitis*. The abnormal secretion is grayish white and not abun-

dant, and has a tendency to adhere to the reddened lid margins and to gather in small masses at the angles, especially the inner one. This clinical manifestation is so constant that it is well-nigh characteristic, and if there is any doubt, the diagnosis is readily established by finding the bacillus in the secretion.

On the other hand the reaction of the conjunctiva to the Morax bacillus is often *acute*, or the inflammation may be confined to the conjunctiva without special participation of the lid angle, or the disease may resemble an acute conjunctival catarrh with swelling. In other words, it may vary from a mild hyperemia of the conjunctiva to a severe mucopurulent conjunctivitis (McKee). Morax-Axenfeld conjunctivitis may attack persons of all ages, but is more common in adults than in young persons. It is widely distributed over the world. It may arise at any season, but is said to be more frequent in summer than during cold weather. McKee, however, found the greatest number of cases in January. Diplobacillary conjunctivitis may be accompanied by a nasal catarrh, and the diplobacilli may be found in the nasal secretion.

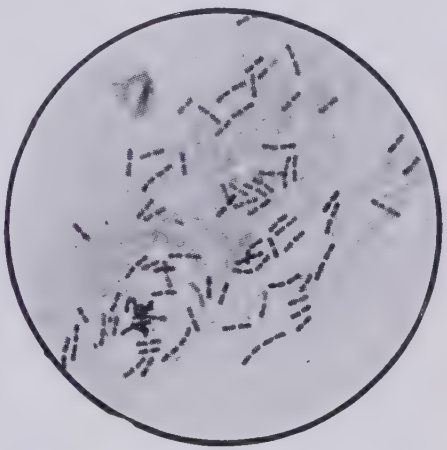


Fig. 106.—The diplobacillus of Morax and Axenfeld (from a preparation by Dr. Harold Gifford).

The disease may last from six weeks to six months, but if improperly treated may remain for much longer periods of time. Its duration is readily shortened by proper treatment. It may be associated with follicular and phlyctenular conjunctivitis, and *corneal complications* may arise, either in the form of superficial infiltrations or deep ulcers, from which the bacillus may be cultivated (*diplobacillary keratitis*). Iritis without corneal implication has been observed (Ernest Thomson). Occasionally these ulcers are complicated by hypopyon and iritis (L. Paul).

Petit has isolated a bacillus resembling the Morax-Axenfeld organism which can produce a conjunctivitis like the one just described. It may also attack the cornea primarily and is capable of originating hypopyon-keratitis. The *Bacillus pyocyaneus* sometimes causes a conjunctivitis which yields to zinc sulphate (Brown Pusey).

Treatment of Conjunctivitis.—(*a*) *Simple Conjunctivitis.*—This consists, first, in search for the cause and the alleviation of associated conditions. The patient must be removed from the influence of dust, cold winds, tobacco smoke, and the like; the under surfaces of the lids should be examined for foreign bodies, and their borders for misplaced

cilia. In the earlier stages cold compresses are agreeable and suitable; sometimes frequent bathings with hot water are more acceptable. At first a solution of boric acid, as collyrium or spray—gr. x to f℥j (0.65 gm. to 30 c.c.) is useful. The eyelids and ciliary margins should be frequently washed with water and Castile soap.

As soon as the discharge becomes mucous or mucopurulent and the velvety opacity of the conjunctiva forms, a stronger solution of boric acid, to which a few grains of common salt may be added, is advisable; and the everted lids may be painted with a solution of nitrate of silver, gr. v–x to f℥j (0.324–0.65 gm. to 30 c.c.), followed by an irrigation with physiologic salt solution. In place of nitrate of silver, protargol (5–20 per cent.) and argyrol (10–25 per cent.) are frequently employed. In mild cases one or two applications a day are sufficient, the drug being dropped into the conjunctival sac and allowed to spread freely over the inflamed membrane. Neosilvol (15 per cent.) may be used with advantage. Argentamin (2–5 per cent.) and largin (10 per cent.) are also recommended. In severe types, with a considerable discharge, bichlorid of mercury (1 : 8000–10,000) and cyanid of mercury (1 : 2000) are valuable collyria.

Other preparations which have found favor are alum, gr. iv–viij to f℥j (0.26–0.52 gm. to 30 c.c.), sulphate of zinc, gr. ij to f℥j (0.13 gm. to 30 c.c.), which may be suitably combined with boric acid, biborate of sodium, gr. iv–viij to f℥j (0.26–0.52 gm. to 30 c.c.), peroxid of hydrogen, Panas' fluid, creolin (1 per cent.), and other antiseptic collyria. Should the thickening of the retrotarsal folds prove stubborn, after the acute symptoms have subsided, these may be touched with an alum crystal or a solution of tannin and glycerin. Atropin is not usually necessary unless a corneal ulcer complicates the affection.

The eyes may be protected by smoked glasses, but in no circumstance should they be bandaged or be covered with poultices of tea-leaves (which of themselves may produce conjunctivitis—"tea-leaf conjunctivitis"), bread and milk, scraped potatoes, and the like. It should be remembered that meddlesome domestic medication of this sort may change a simple conjunctivitis into a serious and purulent inflammation.

At the outset a laxative, followed by full doses of quinin, is indicated; any associated disease of which the conjunctivitis may be a symptom, *e. g.*, rhinitis, requires the usual treatment. Proper hygiene, fresh air, strict cleanliness, and protection from contaminated towels, etc., are evident indications.

(*b*) *Acute Contagious Conjunctivitis.*—The treatment does not differ from that which has already been described in connection with simple conjunctivitis. Weeks recommends bichlorid of mercury (1 : 10,000) as a collyrium. Usually argyrol and protargol are prescribed, and they may act efficiently, but it is a mistake to permit patients themselves to use these preparations for long periods of time. As Risley has aptly said, they may originate a new set of conjunctival catarrhs, and give rise to a troublesome, tumid, hyperemic condition of the conjunctiva;

also they may stain the membrane. Applications of nitrate of silver, in the usual manner, to the everted lids are useful. Iced compresses afford relief during the height of the affection. In place of the ordinary collyria, chlorid of zinc, 1 grain (0.065 gm.) to the ounce (30 c.c.), is highly recommended; sulphate of zinc, gr. ij to f℥j (0.13 gm. to 30 c.c.), is also valuable.

(*c*) *Pneumococcus Conjunctivitis.*—In this affection the treatment already described in the preceding paragraphs is efficient. Sulphate of zinc drops are useful; ethylhydrocuprein may be applied in the usual manner to the everted lids in a 2 per cent. solution or be used as a collyrium ($\frac{1}{4}$–1 per cent.). Mercurophen (1 : 10,000) achieves excellent results.

(*d*) *Influenza Bacillus Conjunctivitis.*—The usual collyria and antiseptics are advisable. Zur Nedden recommends nitrate of silver (1.5–2 per cent.), oxycyanid of mercury (1 : 1500), and iced compresses.

(*e*) *Diplobacillus Conjunctivitis.*—In this disease the preparations of zinc are practically specifics. They may be employed in the form of the sulphate (0.5–1.5 per cent.) or the chlorid (0.2 per cent.). A useful method is to paint the everted lids with 2 to 3 per cent. solution of sulphate of zinc once a day and provide the patient with 0.5 per cent. solution to be used freely and frequently. Nitrate of silver, argyrol and protargol, in contradistinction to their valuable action in other varieties of conjunctivitis, are of no use. Todd recommended sozoiodolate of zinc (1–2 per cent.) and ichthyol ointment (10 per cent.).

Catarrhal Epidemic Conjunctivitis (*Catarrh with Swelling; Epidemic Catarrh*).—Certain systematic writers, notably Saemisch and Schmidt-Rimpler, although they considered this disease to be a form of acute conjunctival catarrh, gave it a separate description on account of certain distinguishing features, namely, swelling not only of the lid margin, but of the entire lid itself, which seems to be increased in volume and is reddened, together with tumefaction, infiltration, and marked hyperemia of the retrotarsal folds. The secretion is considerable in quantity, mucopurulent in character, and often mixed with small fibrinous masses. Both eyes are usually affected, and persons of any age may be attacked. It has appeared with notable frequency among scrofulous children, especially if they are also the subjects of exanthematous disease of the face. The secretion should be considered as distinctly communicable, and the disease has appeared in the form of small epidemics. Sometimes it is a sequel of influenza. In the secretion, pneumococci, staphylococci, and streptococci have been found. The treatment should include thorough irrigation of the conjunctival sac with bichlorid of mercury (1 : 8000), cyanid of mercury (1 : 5000), mercurophen (1 : 8000) or a saturated solution of boric acid. The swollen and inflamed retrotarsal folds may be treated with applications of 1 to 2 per cent. solutions of nitrate of silver in the usual manner, or in their place protargol and argyrol may be employed.

Exanthematous Conjunctivitis.—This form of conjunctivitis has been briefly referred to in connection with catarrhs of the conjunc-

tiva, and is particularly noteworthy as part of the manifestations of measles, scarlet fever, and smallpox. The disease may not only accompany these exanthems, but often arises prior to their eruption. Smallpox pustules sometimes develop on the conjunctiva. Occasionally the conjunctivitis of measles assumes a very severe type, so severe that it may resemble a blennorrhea, and Fuchs has noted under these conditions a suppurative inflammation of the Meibomian glands. The investigations of Schottelius indicated that staphylococci and streptococci are active in the conjunctivitis of measles, streptococci being especially common in fatal cases.

Among the chronic exanthemata of the conjunctiva, *acne rosacea* is important (see also page 207), and in this disease minute nodules may form with marked irritation near the limbus. The conjunctiva may also be implicated in pityriasis, psoriasis, and herpes iris. Its involvement in syphilis, pemphigus, lupus, and lepra is elsewhere described.

Treatment.—The treatment of exanthematous conjunctivitis does not differ from that of the catarrhal form of the affection and its varieties. Fuchs recommends the local application of calomel as especially valuable in acne rosacea of the conjunctiva.

Unusual Forms of Conjunctivitis.—A very rare form of conjunctivitis or conjunctival disease is *squirrel plague conjunctivitis*, described by Derrick Vail and Robert Sattler, due to an infection with the *Bacillus tularense*, identified by McCoy and Chapin as the germ of squirrel plague. In Vail's patient the symptoms were: intense chemosis, mucoid secretion, enlarged preauricular glands, and deep yellow necrotic ulcers in the palpebral conjunctiva. *Samoan Conjunctivitis*, according to Eby, is acutely infectious and is characterized by its rapid onset, the intensity of the conjunctival inflammation, pain and dread of light. The discharge quickly becomes purulent and destructive corneal involvement may be a complication. *Infectious necrotic conjunctivitis* according to Pascheff, begins with constitutional symptoms. There are enlargement of the preauricular and submaxillary glands, reddening of the tarsal conjunctiva and infiltration of the retrotarsal folds, with formation of white spots which break down into ulcers. The disease is said by Pascheff to last from one to three weeks. He failed to identify the bacteria which were found. The similarity of the manifestations of this disease and those of squirrel plague conjunctivitis is suggestive.

James M. Patton and S. R. Gifford have studied a conjunctival affection to which they give the name *agricultural conjunctivitis*. The condition is characterized by enormous swelling of the lids, swelling of the adjoining lymph-nodes, some superficial necrosis of the skin of the lids, and pseudomembranous deposit on the conjunctiva. The patients came from farming districts. Bacteriologic investigation showed a mixed infection—streptococcus, staphylococcus, xerosis bacillus, and large Gram-positive anaërobic bacilli.

Purulent conjunctivitis (*acute blennorrhea of the conjunctiva*)

occurs in three specific forms: in the newborn (*ophthalmia neonatorum*), in young girls (occasionally boys), and in adults (*gonorrheal conjunctivitis* or *ophthalmia*).

Conjunctivitis Neonatorum (*Ophthalmia Neonatorum*).—This is an inflammation of the conjunctiva, characterized, in its usual form, by great swelling of the lids, tumefaction of the palpebral conjunctiva, serous infiltration of the bulbar conjunctiva, and the free secretion of pus.

Causes.—The affection is most frequently caused by the introduction into the eye of infecting material from some portion of the genito-urinary tract of the mother at the time of or shortly after birth. The majority of cases (60–70 per cent.) are associated with a special micro-organism—the *gonococcus of Neisser*. Exceptionally, inoculation occurs *in utero*, owing to the penetrating power of the gonococcus or to infection after rupture of the membranes (*antepartum conjunctivitis*).

Many observers have demonstrated that ophthalmia neonatorum is not always gonorrheal in origin, but may be produced by various kinds of micro-organisms—pneumococcus, streptococcus, diplobacillus, Bacterium coli, and Staphylococcus albus, aureus, and citreus. It is possible that some forms of conjunctivitis neonatorum are really types of influenza bacillus conjunctivitis; the acute purulent conjunctivitis due to the Micrococcus catarrhalis has been referred to. According to Stephenson, next to the gonococcus the pneumococcus is the commonest organism found with ophthalmia neonatorum. *Inclusion blennorrhea of the newborn* is referred to elsewhere (see page 223).

Inasmuch as the gonococcus is not invariably present, two forms of the disease have been distinguished—a severe type, caused by the gonococcus, with a tendency to increase in severity and invade the cornea; and a milder type, non-specific, with a tendency to recover. Hence, a virulent vaginal discharge is not necessary to produce this condition, except in intense degree, and it probably may arise from the contamination of any mucopurluent discharge during birth, and from injudicious intravaginal antisepsis with strong solutions of mercuric chlorid. Careless bathing of the child after birth and the use of soiled towels and sponges are fruitful sources of infection. Contact with the lochial discharge may originate the disorder, although inoculation with healthy lochia has failed to produce the disease.

Infection is more likely to occur in face presentations and during retarded labors. Boys are attacked more frequently than girls. The disease is said to be more common during summer months in cold climates; in hot countries, during the spring and autumn. Ophthalmia neonatorum is more common among children illegitimately born than among those of legitimate birth.

Symptoms.—Conjunctivitis neonatorum usually begins on the third day after birth, but may set in as early as from twelve to forty-eight hours after inoculation, or, where it is the result of a secondary infection from soiled fingers, sponges, or cloths, be delayed to a much later date. Late gonococcic infections have been explained by Credé-

Hörder on the assumption that the Neisser organisms do not find their way directly into the conjunctival sac during birth, but may be hidden, for example, in Meibomian glands. Non-gonorrheal cases do not usually arise until after the fifth to the seventh day. In conjunctivitis neonatorum almost always both eyes are attacked, the one being earlier and frequently more decidedly affected than its fellow.

Four stages of the disease are common, but as these vary in different cases, and more or less rapidly shade one into the other, no very sharp lines need be drawn.

A slight redness of the conjunctiva, with a trifling discharge in the corner of the eye, is rapidly succeeded by great, cushion-like swelling of the lids, with intense chemosis and congestion of the conjunctiva, accompanied by severe pain and discharge. The surface of the swollen lid is hot, dusky red, and tense; the upper lid overhangs the lower, and

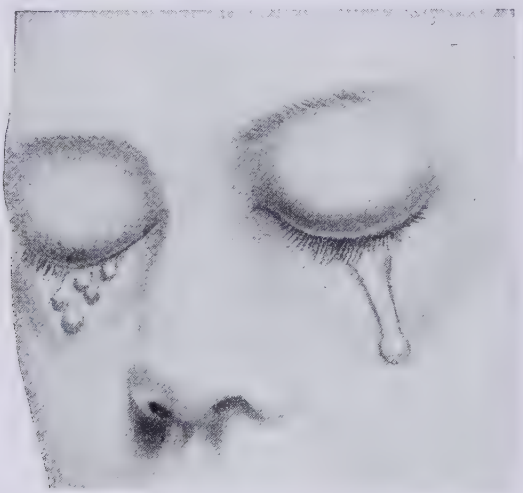


Fig. 107.—Conjunctivitis neonatorum (from a patient in the Philadelphia General Hospital).

at first can be everted only with difficulty. The discharge, which in the beginning is slightly turbid, soon changes to a yellow or greenish-yellow pus, and is secreted in great quantities.

If the lids are everted during the first day or two of the disease, the conjunctiva will be found to be swollen, red, and velvety, and that upon the eyeball intensely injected; upon the surface easily detached flakes of lymph are found; later, the conjunctiva becomes rough and of a dark red color, spots of ecchymosis appear, or it is succulent and bleeds easily. Marked chemosis and infiltration of the ocular conjunctiva succeed, forming a hard rim; at the bottom of the crater-like pit thus produced the cornea may be seen. The thick, cream-like discharge increases, and either flows out from beneath the overhanging upper lid on to the cheek or is packed up in the conjunctival culdesac (Fig. 107). Sometimes false membrane forms and covers the tarsal

conjunctiva; indeed, the appearances may be exactly like those of a *membranous conjunctivitis.*

The lids now may lose much of their tense character, and can be more easily everted; the conjunctiva is puckered into folds and papilla-like elevations, and the discharge contains an admixture of blood and serum. Gradually the disease declines, and in from six to eight weeks the discharge ceases. The relaxed palpebral conjunctiva is thick and granular, looking like the granulation tissue which surrounds wounds. The ocular conjunctiva is also thickened, and positive cicatricial changes may remain.

The chief risk is destruction of the vitality of the cornea, the danger of which is materially increased if this membrane becomes lusterless, dull, and hazy within the first day or two of the disease, and the gonococcus is freely present in the discharge. Frequently small oval ulcers form near the limbus, either transparent or surrounded by an area of cloudy infiltration, which rapidly increase in size; or larger areas of ulceration develop in a more central situation. In many mild cases the cornea escapes without harm. The changes which take place in the cornea are due in part to strangulation of its nutrient vessels by the swollen tissue, but largely to direct infection by the discharge. Corneal lesions do not usually occur if the discharge is free from gonococci.

M. S. Mayou classifies the corneal ulcers of ophthalmia neonatorum as *puncture ulcers*, which may penetrate the cornea and cause anterior polar cataract; large *infiltrated ulcers*, which, if they perforate the cornea, produce adherent leukoma; and *sloughing ulcers* which involve the whole cornea.

The result of perforation will depend upon the amount and character of the destruction of the corneal tissue. If the ulcer is central and perforates, the aqueous humor escapes, the lens is pressed forward against the posterior surface of the cornea, and the opening becomes closed with lymph. Restoration of the anterior chamber follows, and the lens returns to its proper position, carrying with it upon the anterior capsule a little mass of lymph. Thus the formation of a *pyramidal* or *anterior polar cataract* results (see page 459). Fuchs maintains that in the formation of opacities following corneal suppuration the lens epithelium is rarefied by destruction of the cells; later there is proliferation of the remaining cells and *capsular cataract* results.

Perforation of an ulcer peripherally situated, especially below, is followed by adhesion of the iris to the opening. The aqueous escapes, and, as the iris and the lens fall forward, the former becomes entangled in the perforation and is fixed by inflammatory exudation. The adhesion is either on the posterior surface or in the cicatrix, and the resulting dense white scar receives the name *adherent leukoma.*

If the region of the scar is bulged forward because it is unable to resist the intra-ocular tension, *anterior staphyloma* results. Extensive sloughing of the corneal tissue, with total prolapse of the iris, matting together of the parts by exudation, and protrusion of the cicatrix constitute a *total anterior staphyloma.*

Finally, perforation may be followed by inflammatory involvement of the ciliary body and choroid, and the rapid destruction of the eye by *panophthalmitis*, or a slower shrinking of the tissues, with *atrophy* of the *bulb*. Dense opacity occasionally appears in the cornea during convalescence, and may go on to ulceration or clear up perfectly. It may arise with great suddenness, and, should it occur in the lower half of the cornea, a deep indentation, owing to the pressure of the margin of the lid, is likely to occur.

The appearance of the conjunctiva differs materially in different cases. Its surface may be covered over not merely by easily detached flakes of lymph, but by a gray, false membrane. More rarely a deep infiltration develops, like that seen in diphtheritic conjunctivitis.

Restlessness, fever, and other constitutional disturbances are sometimes present, and synovitis of the knee and wrists may arise, of the same character as arthritis occurring in adults during gonorrhea. Rhinitis, infection of the lacrimal gland, meningitis, endocarditis, and general septicemia have been reported as complications of ophthalmia neonatorum. An unusual complication is gangrene of the lids. In rare instances pneumococcus conjunctivitis neonatorum may be associated in the second week with inflammation of the knee-joint (Stephenson).

Conjunctivitis neonatorum does not always follow this course, because the term is made to include affections of the conjunctiva in the newborn other than the types just described—mild catarrhal conjunctivitis, hyperemias, and that variety which, according to Noyes, presents the character of a granular, rather than of a purulent, conjunctivitis, and which may continue for weeks without danger of corneal complication. Occasionally a gonococcal conjunctivitis pursues the course of a simple conjunctival catarrh (Groenouw). Furthermore, as Saemisch has said, purulent conjunctivitis may develop in newborn children not due to the gonococcus (see also page 215), but caused either by other virulent bacteria or by non-bacterial agents. Necessarily the manifestations are less violent than those of gonococcal origin, and for these varieties the name *acute blennorrhagic conjunctivitis neonatorum* has been suggested.

Some hyperemia of the conjunctiva, with a little yellowish discharge in the corners of the eye and slight swelling of the lower lid, is common in babies for a few days after birth.

Diagnosis.—The onset and character of the typical disease, its symptoms and course, render a mistake in regard to its nature practically impossible. Close attention should be given to what at first appears to be a trivial inflammation in the eyes of a newborn child, because a virulent and destructive inflammation may follow with great rapidity. Bacteriologic examination of the secretion is essential, which should include not only smears but also cultures, and the findings will determine the true character of the disease. Recognition of the gonococcus usually is not difficult, but the danger of mistaking the Micrococcus catarrhalis for the Neisser organism must not be disregarded (page 207).

Prognosis.—This is always grave in gonorrheal cases, but with competent medical attendance, *if the eyes are seen while the corneas are still clear*, except in diphtheritic types, in those with inherent malignancy (Randall), or where depreciation of nutrition or intercurrent illness diminish the resisting power of the child, the majority of cases should be brought to a successful termination. Hence the attendants of newborn children should be compelled to seek medical advice as soon as conjunctival trouble appears, for delayed or improper treatment means sloughing of the cornea, when no form of medication can do more than relieve the violence of the inflammation, which, after it subsides, leaves the child with sight hopelessly marred, perhaps destroyed. The prognosis of the mild types is favorable.

Prophylaxis.—The present high standard of scientific midwifery includes such cautious antisepsis prior to and during labor that the risk of contamination is distinctly less than in former times, but still some preventive method should be employed.

The eyes of those children who have passed through a birth-canal known to be infected, or from which the suspicion of infection cannot positively be eliminated prior to birth, should be treated according to the method of Credé, which is as follows: As soon as the head is born the lids are carefully cleansed, parted, and 2 drops of a 2 per cent. solution of nitrate of silver are instilled into each conjunctival sac. Small cold compresses are then laid upon the lids and renewed at suitable intervals. Occasionally severe reaction follows—conjunctival hyperemia or catarrh (the so-called "silver catarrh"), and even hemorrhage from the conjunctiva and corneal haze. Hence it is not always necessary to employ a 2 per cent. solution of nitrate of silver, inasmuch as a 1 per cent. solution will be of sufficient strength. Wherever infection, or the suspicion of infection, can be *positively* excluded, milder measures —for example, washing the eyes and flushing them with a saturated boric acid solution—are sufficient. Other materials recommended are aqua chlorini (Schmidt-Rimpler), sophol (5 per cent.) (von Herff, R. M. Williams), bichlorid of mercury (1 : 5000), carbolic acid (1 per cent.), and the newer silver salts, especially argyrol (25 per cent.) and protargol (10 per cent.). The last-named remedies are not to be trusted in the management of eyes which have been exposed to gonorrheal infection. The value of Credé's method is so firmly established that it should not be neglected if the birth-canal is known to be infected with gonorrhea, or if the suspicion of infection cannot be excluded. In its place a 5 per cent. solution of sophol has been recommended, but there is no satisfactory evidence that it should, as has been advised by some surgeons, supplant nitrate of silver. With this drug the author has had no experience. The hands of the mother, nurse, and child should be searched for sources of infection, and if gonorrhea is known to exist in the mother the child should be isolated.

Conjunctivitis neonatorum should be listed as a reportable disease, and laws should be enacted to this effect, and these laws should be rigidly enforced, and it should be possible to provide the affected children with

expert attention at their homes or, even better, with hospital accommodations. The distribution by health boards of circulars of advice to midwives and mothers and of tubes containing the chosen prophylactic to those who are qualified to use it (preferably a 1 per cent. solution of nitrate of silver) should be required. Happily, there has been a real advance in the methods, legal and otherwise, for preventing ophthalmia neonatorum, but much work remains to be done. As ophthalmia neonatorum is the cause of fully 8 per cent. of blindness in this country, efforts to check the ravages of this disease cannot too strongly be advocated.

Treatment.—If the type is mild, the remedies suitable for simple conjunctivitis are indicated; if severe, three conditions demand attention: the swelling of the lids, the state of the conjunctiva, and the corneal complications.

1. During the period when the lids are tense and the secretion lacking in its later creamy character, in addition to absolute cleanliness, local application of cold is a useful agent in a certain number of cases.

This should be applied in the following manner: Upon a block of ice square compresses of gauze are laid which, in turn, are placed upon the swollen lids, and as frequently changed as may be needful to keep up a uniform cold impression. This is far preferable to the use of small bladders containing crushed ice; indeed, the use of ice is not advisable. The duration of cold applications must vary according to the severity of the case. Sometimes they may be used almost continuously, and sometimes every three or four hours for twenty minutes at a time. Standish and other surgeons deny the value of cold in this disease, believing that it adds to the danger of corneal complications. This is entirely contrary to the author's experience.

It requires a good deal of experience to know when to use and when not to use cold, and not all cases are suited to its application. Hot fomentations have been advised, especially where corneal complications exist, or the surface of the conjunctiva is covered with a gray film. These are applied with squares of antiseptic gauze wrung out in carbolized water of a temperature of 120° F., and frequently changed. The author, basing his opinion on a large experience in the Philadelphia General Hospital, doubts their value.

2. Constant removal of the discharge must be practised.

The lids should be gently separated, the tenacious secretion wiped away with bits of moistened lint or absorbent cotton, and the conjunctival sac freely irrigated with an antiseptic fluid. For this purpose a saturated solution of boric acid (which is feebly antiseptic, but very cleansing and slightly astringent) or one of corrosive sublimate, 1 grain to 1 pint (0.065 gm. to 480 c.c.) (strong solutions should not be used because they may injure the corneal epithelium and cause ulceration), may be employed. Special and ingenious forms of lid irrigators have been devised, but are unnecessary and often harmful. The cleansing process must be repeated at least every hour, day and night, and, if necessary, much more frequently; but all manipulations must be most

gentle and great care not to injure the delicate structures of the eye must be exercised. Irrigations with a solution of magnesium sulphate are highly recommended by Kirkpatrick (see page 225).

3. The application of one of the salts of silver.

Until comparatively recent times nitrate of silver was almost universally employed, a drug combining the properties of an astringent and a superficial caustic. Its germicidal power, however, is not of much avail, as this is quickly impaired by contact with the tissue proteins. Once a day the palpebral conjunctiva and retrotarsal folds should be brushed over with a solution, 10 grains (0.65 gm.) to the ounce (30 c.c.), their surfaces first having been carefully freed from any adherent discharge, and afterward all excess of the drug washed away with a solution of common salt, and this washing continued until a clean red surface is secured, when the lids may be returned to their proper position, their margins greased with vaselin, and some of the lubricant introduced within the conjunctival culdesac. Ulceration of the cornea does not alter the treatment described. As long as the discharge is abundant the use of the caustic is indicated, and it may be employed from the very beginning of the disease unless the conjunctiva is covered by a false membrane, which would prevent its access to the conjunctival folds.

Within the last few years protargol and argyrol have largely replaced nitrate of silver in the treatment of ophthalmia neonatorum in the practice of many surgeons. Standish recommends the following routine treatment or "immersion method." The edges of the lids are washed with a solution of boric acid once in half an hour, and they are anointed with vaselin to prevent them from sticking together. A solution of protargol or argyrol is instilled freely between the lids at intervals, that is, every hour or once in four hours according to the conditions. This fluid sinks to the bottom of the culdesac and floats to the surface the pus and mucus, which can readily be removed with a very slight amount of manipulation. Protargol is used in strengths varying from 10 to 20 per cent., the 10 per cent. solution yielding the better results. Of argyrol, a 25 per cent. solution is satisfactory. Henry D. Bruns prefers a 10 per cent. solution of argyrol to be used freely every half-hour until pus secretion is checked. Then he applies in the usual manner nitrate of silver (0.2–1.0 per cent.) once a day. Many other surgeons, both here and abroad, employ these drugs in a similar manner. According to Stephenson, in gonorrheal ophthalmia neonatorum a 25 per cent. solution of argyrol painted once or twice a day over the conjunctiva, exposed for that purpose by eversion of the lids and carefully freed from adherent discharge, with the frequent use of instillation of the 25 per cent. or of a weaker solution, represents a method of treatment more promptly efficacious than any other with which he is acquainted.

The author's experience is in accord with the good results ascribed to the methods of using argyrol just described, except that in a certain number of cases the argyrol treatment alone is not sufficient, and nitrate of silver must be used, especially, as Bruns recommends, in addi-

tion to or after the use of argyrol. In other words, the author, because of a large experience in the ophthalmic wards of the Philadelphia General Hospital, is unconvinced that nitrate of silver, properly applied (p. 221), has ceased to be a valuable remedy in ophthalmia neonatorum.[1] Protargol as compared with argyrol is more irritating (although not markedly so) and does appear to possess special advantages. It is usually stated that argyrol is without germicidal properties, but a research by Lancaster indicates that it is not without potency in this respect, and Robert Cheney has reached similar conclusions provided the bacteria are well separated and do not exist in clumps or masses. Its chief virtues from the clinical standpoint, however, are its freedom from irritating qualities, its marked detergent effects, and its ability to penetrate between the folds of swollen conjunctiva and liberate the secretion.

At the first appearance of corneal haze 1 drop of a 0.5 per cent. solution of atropin should be dropped into the eye two or three times daily. During corneal complications Darier recommends a collyrium containing dionin, pilocarpin, and cyanid of mercury.

Persistent swelling of the conjunctiva is sometimes treated by scarification. Division of the outer commissure to relieve pressure is not suited to young infants, although it may be indicated in adults.

If one eye alone is affected, suitable protection for the sound eye should be provided. This may be accomplished by antiseptic bandaging of the uninflamed organ (Buller's shield is difficult of application in infants).

The attendants must be impressed with the fact that upon their faithful carrying out of directions, and upon their unremitting care, much, if not all, of the hope of bringing the case to a successful termination depends. The attendants must further be impressed with the contagious nature of the pus; all bits of rag and pledgets of lint used in the treatment must be destroyed, and after each treatment the hands of those engaged in it must be thoroughly washed and disinfected with a solution of bichlorid of mercury.

Many other remedies have been used in the treatment of conjunctivitis neonatorum; for example, those mentioned on page 212 and carbolic acid (0.5 to 1 per cent.), iodoform and iodoform ointment (4 per cent.), aqua chlorini, cyanid of mercury (1 : 1500), permanganate of potassium (1 : 5000), used in copious irrigations, formaldehyd (1 : 5000), and argentamin (2 per cent.). Darier suggests a 3 per cent. solution of ichthargan if protargol loses its effect. Blenolenicet salve has been recommended (Adams, Scheuermann), and sophol in 5 per cent. solution (von Herff). These remedies do not seem to possess virtues which justify their use in place of those which have been more fully described.

[1] The author is well aware that many surgeons deprecate the use of nitrate of silver in these circumstances. George Derby, for example, thinks the reaction in gonorrheal conjunctivitis is too great to permit its use and that its bactericidal power cannot assail the deep-seated gonococci. He prefers mild remedies, *e. g.*, argyrol. E. B. Heckel reports excellent results from constant irrigation alone with iced physiologic salt solution.

Inclusion Blennorrhea of the Newborn.—In certain varieties of conjunctivitis neonatorum, usually non-gonorrheal and for the most part benign in nature, the Giemsa stain will reveal in the secretion "epithelial inclusions," that is, clusters of small granules, colored violet or dark blue, which cap the nucleus like a cowl. According to Axenfeld, this "inclusion blennorrhea" is frequently unilateral, the retrotarsal fold is tumid, and if the disease lingers long a granular condition of the conjunctiva supervenes. After its subsidence sometimes delicate cicatrical tissue remains. Corneal complications are not in evidence. Whether these "inclusions" are the cause of this disease is not known (see also page 238).

Purulent Conjunctivitis in Young Girls.—Occasionally young girls are the subjects of vaginitis, which in severe forms is associated with a purulent discharge, and in hospitals and asylums has occasionally assumed the form of an epidemic among the inmates. In a certain percentage of these cases gonococci are present in the discharge, and the disease may be conveyed to the eye by the fingers, or gain entrance into the conjunctival sac from discharge adherent to bed-linen, sponges, etc. There results a purulent conjunctivitis, with symptoms closely resembling those of ophthalmia neonatorum, although usually the manifestations are less violent, and the corneal complications less likely to occur than in the gonorrheal conjunctivitis of adults. To this disease the name *ophthalmoblennorrhea*, or *gonoblennorrhea, of young girls* has been given. The treatment should in all respects conform to that which has been described in connection with ophthalmia neonatorum. If the disease is properly treated the prognosis is good.

Gonorrheal conjunctivitis of adults (*purulent ophthalmia; acute blennorrhea in adults*) usually can be traced to the source of infection which arises from an acute gonorrhea or a gleet, by contact with soiled fingers or linen, or from an eye affected with this form of conjunctivitis. Considering the frequency of gonorrhea this ocular complication is not a very common condition, occurring according to White, quoted by Duane, about once in eight hundred cases of gonorrhea. If all sources of infection are considered, however, it is probable that its incidence is greater than these figures indicate.

The same micro-organism described in connection with gonorrheal ophthalmia neonatorum is active in gonorrheal conjunctivitis, the diplococci being found within the cells; later they penetrate the epithelium and enter the lymph spaces in the subconjunctival tissue.

Symptoms.—The first symptoms appear from twelve to forty-eight hours after inoculation, and resemble those already recorded in connection with the same disease occurring in the newborn (see page 216).

The vitality of the cornea is in constant danger, and extensive involvement of this membrane may arise during the height of the attack or later, and when convalescence apparently is established. This involvement consists in ulcers, small and large, either central or peripheral; in the latter position they often exist as grooved rings or small clean-cut lesions without infiltration, hidden by the swelling of the sur-

rounding conjunctiva, and very prone to perforate; or they may coalesce and form a *ring abscess*. The ulcers of gonorrheal conjunctivitis are apt to be elaborate and infiltrated or sloughing. A more or less dense opacity may follow ulceration or arise independently of this condition.

If perforation occurs, all the phenomena described on page 217 develop and even without perforation, iritis, cyclitis, and disease of the deeper structures of the eye may supervene and defeat the possibility of obtaining good vision.

Gonorrheal conjunctivitis reaches its climax in about ten days and then gradually subsides in from one to two months; or it may pass into a chronic type and be one of the forms of *chronic blennorrhea*, in which there is general redness of the palpebral conjunctiva, with hypertrophy of its superficial layers and some thickening of the papillæ.

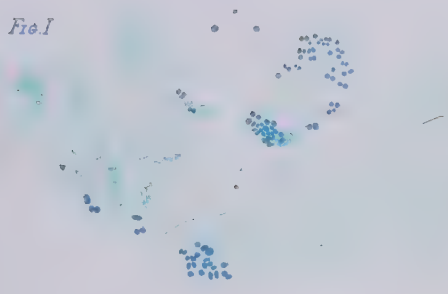
Diagnosis.—This is readily made from the history of the case, and, above all, by an examination of the secretion for the gonococci in stained smears and by cultures.

Prognosis.—The prognosis is *always* grave, much more so than in conjunctivitis neonatorum. The subject of fully developed gonorrheal conjunctivitis comparatively rarely recovers without some corneal involvement, and only too often the eye is hopelessly marred. It may be stated, however, that the prognosis is better now as the result of improved methods of treatment than in former times. It is probable that certain cases diagnosticated as true gonorrheal conjunctivitis which have yielded with astonishing rapidity to therapeutic measures have not been due to a Neisserian infection. The difficulty of distinguishing between the gonococcus and the Micrococcus catarrhalis has been discussed (page 207). Arthritis, endocarditis, and septicemia may arise as complications (see also page 218).

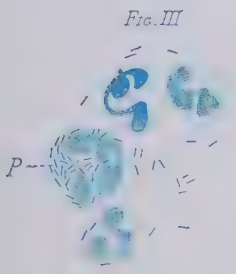
Treatment.—This includes the same remedies described in connection with ophthalmia neonatorum (see page 220), but requires certain modifications suggested by the adult age of the majority of the patients.

If the swelling of the lids is so great that their pressure threatens to destroy the cornea, the outer canthus may be divided (canthotomy). This acts in a twofold manner, by relieving pressure and by depleting the engorgement. The incision should be made with a scalpel, cutting the tissues from without down to the bone as far as the margin of the orbit, but leaving the conjunctiva uninjured. Repeated incisions of the hard rim of chemotic conjunctiva which surrounds the cornea will also relieve pressure, and in some circumstances is a most useful procedure. In desperate cases some operators (Critchett, Fuchs) have not hesitated to split the lid vertically and stitch the divided portions to the brow, restoring them by a plastic operation after the disease has subsided. Cold may be applied with compresses in the manner already described, or continuously with the aid of Leiter's tubes. Certain experienced surgeons, as has already been mentioned in connection with ophthalmia neonatorum, doubt the value of cold applications. The author believes that in gonorrheal conjunctivitis in adults, during the early stage, cold is not only most agreeable to the patient, relieving pain and

PLATE II.







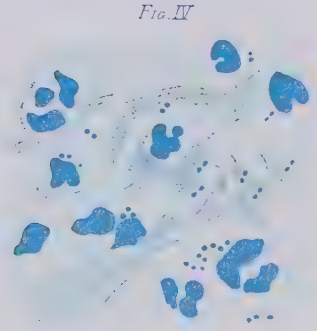


FIG. I.—Discharge from right eye in a case of purulent conjunctivitis; gonococci numerous in cells (Stephenson).

FIG. II.—Bacillus of Weeks in pure culture (from a photograph) (Weeks).

FIG. III.—Conjunctival secretion from acute contagious conjunctivitis; polynuclear leukocytes with the bacillus of Weeks; *P*, Phagocyte containing bacillus of Weeks; immers. $\frac{1}{12}$, oc. iii (Morax).

FIG. IV.—Secretion from a case of conjunctivitis, showing pneumococci; immers. $\frac{1}{12}$, oc. iii (Morax).

irritation, but of distinct value in checking the inflammatory process and the movement of the bacteria. Not all cases are suited to cold, and it is not always necessary to employ it continuously; it may be used for periods of twenty minutes to half an hour every three or four hours.

Local applications include the antiseptic lotions previously recommended (see page 220), in addition to which may be mentioned a drug which the author has often employed in the wards of the Philadelphia General Hospital with success, namely, permangante of potassium, 1 : 2000–5000, used copiously, a pint at a time, in continuous irrigation after the manner of Kalt. These irrigations should be performed three or four times a day, according to the severity of the case and the quantity of the discharge. Nitrate of silver should be used in the manner described on page 220. In place of this drug argyrol and protargol have been much employed. According to Standish, protargol seems to act somewhat better in the gonorrheal conjunctivitis of adults than argyrol. These drugs should be applied in the manner already described, that is, by the immersion method (see page 221). In the opinion of the author, while argyrol may be employed in the treatment of gonorrheal conjunctivis, it should not be used to the exclusion of nitrate of silver, which, he is satisfied, will always hold a high position in the therapy of this disease in adults. Kirkpatrick recommends most highly frequent irrigations of the conjunctival sac with a hypertonic solution of magnesium sulphate (40 grains to the ounce—to saturation). In the ophthalmic hospital at Madras between the irrigations nitrate of silver is used; excellent results have been reported. With subconjunctival injections in the treatment of serious cases of gonorrheal ophthalmia, commended by Hirsch, who uses a solution of oxycyanid of mercury, 1:5000, to which acoin is added, the author has had no experience. Neither has he faith in the value of peroxid of hydrogen diluted to 3 per cent., which has been recommended in the treatment of this disease, nor has he had an opportunity of testing the value of blenolenicet ointment. Treatment with antigonococcus serum does not appear to have been satisfactory.

On the appearance of any of the types of *corneal ulceration* atropin drops should be instilled with sufficient frequency to maintain mydriasis and subdue ciliary hyperemia; indeed, it is a wise precaution to employ this mydriatic from the beginning of the disease. Eserin has also been recommended, or the combined action of eserin and atropin, obtained by using the former drug during the daytime and the latter at night. In the majority of instances atropin will secure the best results. Iodoform freely dusted upon the ulcer is of service; also the other measures recommended for the control of infected corneal ulcers (see page 279). Dionin is advised by Darier.

If perforation has taken place, excision of the prolapsed iris, sometimes advocated, is not without danger, as this procedure may open a way for the entrance of infecting material to the deeper structures of the eye. The final outcome of the case depends upon the extent of corneal involvement and the ultimate treatment of the remaining

leukoma, staphyloma, or shrunken ball requires, according to circumstances, iridectomy, abscission, evisceration, or enucleation.

Often the patients are debilitated, and supporting treatment is indicated, namely, quinin, iron, and strychnin. If there is constipation, calomel and saline laxatives should be administered. The pain, which is often severe, may be controlled by morphin or opium. In place of morphin codein may be employed. It is a mistake, in the serious forms of this disease, to depend alone upon local measures.

The treatment of a *chronic conjunctivitis*, the sequel of an acute attack, depends upon the degree of thickening in the mucous membrane, but is usually best managed by careful exposure of the thickened conjunctiva and applications of nitrate of silver, tannin and glycerin, and the occasional use of an alum or sulphate of copper stick. A collyrium of boric acid, bichlorid of mercury, or sulphate of zinc may be used.

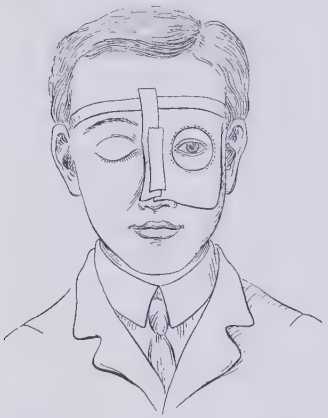


FIG. 108.—Application of Buller's shield.

Prophlyaxis.—Patients suffering from gonorrhea should be warned not only of the great danger of infecting their own eyes, but the eyes of those around them. Inasmuch as a very minute quantity of urethral discharge, and even where this is the product of a chronic disease—gleet, for example—may produce acute conjunctivitis, these precautions should be emphasized.

As usually one eye alone is affected, it is matter of great importance to secure the other eye from contact with the secretions. This may be done by sealing it with an antiseptic bandage, the edges of which are made tight by fastening along them strips of gauze painted with flexible collodion, or by the application of Buller's shield. The latter consists of a watch-glass fitted in a square piece of rubber adhesive plaster, which is carefully applied to the brow, temple, lower margin of the orbit and nose, and should be secured with additional strips to prevent the entrance of discharge.[1] The inner margin should be sealed with collodion, as a contamination is most likely to occur at this point, and inefficient application increases, rather than diminishes, the danger.

All the precautions which have been urged with regard to the care of conjunctivitis neonatorum apply with equal and even greater force to the present disease. In a number of instances the eyes of the surgeon or nurse in attendance have been infected.

Metastatic Gonorrheal Conjunctivitis[2] (*Gonorrheal Epibulbar*

[1] Care should be taken to provide a watch-glass of the ordinary form, not one with a concave center.

[2] Some surgeons apply the name *gonorrheal ophthalmia* to this affection, and reserve the term *gonorrheal conjunctivitis* for the disease which is caused by a specific urethral discharge.

Conjunctivitis—Heerfordt).—This is an inflammation of the mucous membrane of the eye due to the gonococcus which is carried from the urethra to the conjunctiva through the medium of the circulation; or it may be caused by the gonotoxin. According to W. Gordon M. Byers, whose definition has just been quoted, it may occur as the initial symptom of a generalized infection, appear simultaneously with the other manifestations—for example, arthritis—follow the outbreak of inflammatory symptoms elsewhere located, or be the only expression of a systemic gonorrhea.

The disease is almost invariably bilateral, is more common in men than women, and resembles a catarrhal conjunctivitis with some swelling of the mucous membrane and redness of the lids. Small ulcers of the cornea may form. According to Heerfordt, it may assume the form of phlyctenular conjunctivitis. Iritis, iridocyclitis, and uveitis may follow this inflammation just as they may be associated with gonorrheal rheumatism. *Endogenous gonorrheal keratitis,* occurring before or after arthritis, has also been described, but the etiologic relation of gonorrhea is doubtful (Elschnig). Relapses of metastatic gonorrheal conjunctivitis occasionally occur; the average duration of the disease is about two weeks. The gonococcus is not present in the secretion of this type of conjunctivitis.

The treatment of this affection demands the same remedies useful in ordinary conjunctivitis. Heerfordt advises massage with an ointment of yellow oxid of mercury and a lotion of copper sulphate (0.25 per cent.).

Non-specific Purulent Conjunctivitis.—Purulent conjunctivitis, not gonorrheal in origin, may be caused by the secretion of diphtheritic conjunctivitis and by trachoma. As has been noted, the manifestations of Koch-Weeks' bacillus conjunctivitis and pneumococcus conjunctivitis are not infrequently so violent and the secretion so profuse and purulent that they might be classified among purulent inflammations of the conjunctiva. (See also page 209.) It should further be remembered that a catarrhal conjunctivitis, by neglect or injudicious external applications—for example, poultices—may be aggravated into an inflammation resembling gonorrheal conjunctivitis. Infection from the nares and pneumatic sinuses can also cause severe inflammation of the conjunctiva, associated with much purulent discharge. The treatment is exactly that which has been described in connection with other forms of acute conjunctivitis. The best results follow the free irrigation of the conjunctival sac with a saturated solution of boric acid or bichlorid of mercury (1 : 10,000), or mercurochrome (1 per cent.), and the use of argyrol, protargol, and nitrate of silver in the usual manner. If properly treated, corneal complications are uncommon.

Croupous or Pseudomembranous Conjunctivitis (*Plastic, Membranous Conjunctivitis*).—Of this disease, two varieties may be considered. The *first* is an inflammation of the conjunctiva, characterized by a soft, usually painless swelling of the lids, a membranous exudation upon, not within, the conjunctiva, and a scanty, seropurulent discharge.

Causes.—The affection in its pure form is rare, the majority of cases occurring in early life—*i. e.*, between first half year and the seventh year. The transmission of the disease from one eye to another has not been established; no definite cause is known, although formerly an endeavor was made to bring it into relation with scrofula and eczema. Patients affected may at the same time be suffering from a croupous inflammation of the respiratory tract. Some authors regard the affection as a mild diphtheria. Non-virulent Löffler bacilli, staphyloccoci, and diplococci have been found in the secretion.

Symptoms.—The symptoms of the first variety of croupous conjunctivitis begin with an acute inflammation of the conjunctiva, succeeded by swelling of the lids, which remain soft and pliant, and usually not painful to the touch. In a few days there is a deposit of false membrane composed of coagulated fibrin, rather translucent and porcelain-like in appearance, beginning upon the retrotarsal folds, coating the inner surfaces of the lids, but not invading the bulbar conjunctiva. It may readily be removed without loss of the conjunctival tissue, and shows beneath a granular and somewhat bleeding surface. It is quickly reproduced. The cornea, except in severe cases, escapes.

Healing takes place in from ten to thirty days, except where the membrane is formed again and again, and the course of the disease may continue for months and even years, constituting the *recurring form of pseudomembranous conjunctivitis*. Elaborate investigation may fail to find an etiologic factor. This was the case in two patients, both girls, whom the author had an opportunity of studying. Such recurring membranous conjunctivitis has been observed apparently as a complication of erythema multiforme and has lasted for years (Stark). Griffin Lewis, in a patient with Vincent's angina, found on the palpebral conjunctiva a membrane exactly like that which existed in the throat. Instead of affecting the entire surface of the palpebral conjunctiva, a membrane may cover a very limited area and recur frequently—circumscribed chronic membranous conjunctivitis according to Martines and Lutz.

Diagnosis.—The disease may be confounded with conjunctivitis neonatorum and diphtheritic conjunctivitis. From the former it is distinguished by the absence of profuse purulent discharge and by the age of the patient; from the latter, by the soft swelling of the lids, the superficial character of the membrane, and absence of virulent Klebs-Löffler bacilli.

Treatment.—This should include the frequent removal of the discharge with a solution of chlorid of sodium or chlorate of potash, and later the cautious use of nitrate of silver (H. Knapp), or of argyrol and protargol. As in many instances it is difficult to exclude diphtheritic infection, the administration of diphtheria antitoxin should be ordered in doubtful cases. Stark obtained the most satisfactory results with a 1 per cent. solution of sulphate of quinin. The application of a 10 per cent. solution of arsphenamin in glycerin is considered of value in the

treatment of Vincent's angina, and it may be assumed would be of use in case the conjunctiva should be attacked.

The *second* variety of membranous conjunctivitis is due to streptococci, is rapid in development, and is associated with swelling of the lids and much discharge, and may quickly destroy the cornea. It occurs in children in association with measles, scarlet fever, and influenza; but, according to Morax, may appear independently of febrile complications and may accompany impetigo. The prognosis is most unfavorable not only in relation to eyesight but also in relation to life. The disease is often mistaken for diphtheritic conjunctivitis (it is sometimes called *streptococcus diphtheria of the conjunctiva*). Microscopic examination will decide the diagnosis. The treatment already detailed is indicated. According to Axenfeld the use of streptococcal serum is advisable.

Membrane-forming conjunctivitis, not strictly classifiable with either of the foregoing types, occurs as an intercurrent condition in several varieties of conjunctivitis—for example, in gonorrheal conjunctivitis, pneumococcus and Weeks' bacillus conjunctivitis, influenza conjunctivitis, trachoma, and exanthematous conjunctivitis. Membrane may also form in the conjunctiva as the result of chemic, mechanic, and thermic irritants. It is especially noteworthy after certain injuries of the conjunctiva—for instance, lime burns.

Diphtheritic Conjunctivitis.—The deep-seated or necrotic variety of this disease is characterized by a board-like, very painful swelling of the lids, a scanty seropurulent or sanious discharge, and exudation within the layers of the tarsal conjunctiva, which spreads to the ocular conjunctiva, and by pressure destroys the nutrition of the cornea.

Causes.—In addition to the Klebs-Löffler bacilli, which cause the disease, other micro-organisms, for example, streptococci, staphylococci, and non-virulent xerosis bacilli, are usually present in the discharge (Uhthoff). The disease, which is communicable, may originate from a similar case, or arise in the course of a purulent conjunctivitis. It has occurred, though rarely, with conjunctivitis neonatorum. At times it appears in connection with eczema of the face and borders of the lid, and is an occasional accompaniment of some acute illness, for example, scarlet fever or measles, the diphtheritic type of the inflammation being ingrafted upon the conjunctiva. The disease has been seen during epidemics of diphtheria, and may be part of a process which passes from the nose or throat to the conjunctiva, or may be due to direct inoculation with the diphtheritic poison.

It is commonest between the ages of two and eight, and is unusual in young infants. In certain localities in the south of France and the north of Germany the disease was formerly frequent. It is usually stated that the disease is comparatively rare in America and England; but Sydney Stephenson records a percentage of 1.25. This author regards conjunctival diphtheria and croupous conjunctivitis as one and the same disease.

Symptoms.—The patches appear in a *discrete* or *confluent* form;

the lids are swollen with a characteristic, painful, board-like hardness. The false membrane is of a dull, grayish appearance, and is torn off with difficulty, and carries with it parts of the conjunctiva. If the process is deep, the subjacent structure is pale, infiltrated, and when cut into may be anemic and lardaceous. If the diphtheritic inflammation has been ingrafted upon a case of purulent conjunctivitis, the abundant secretion ceases, or becomes irritating and sanious.

Sloughing of the cornea is almost inevitable in severe cases, and rapid destruction of this membrane may take place in twenty-four hours; even in mild cases ulcers may be expected.

Restlessness, fever, alimentary derangements, and nervous phenomena are usual constitutional disturbances, and the disease may be followed by loss of knee-jerk and paresis of various parts of the body. Albumin may be present in the urine, and occasionally diphtheritic conjunctivitis proves to be fatal (Stephenson). Paralysis of accommodation is a rare sequel.

Diagnosis and Prognosis.—This disorder is distinguished from the previous disease by the characteristic board-like infiltration of the lids, by the situation of the membranous exudation within the tissue itself and by the bacteriologic examination, and has nothing in common with the flakes of false membrane sometimes seen in purulent conjunctivitis. The prognosis, as may have been inferred, is unfavorable, although by means of modern treatment more cases are cured without impairment of vision than formerly was possible. Occasionally, even if the diphtheritic membrane is only slightly developed, rapid necrosis of the cornea ensues.

Treatment.—The eyes should be frequently cleansed with warm boric acid solution or bichlorid of mercury solution (1 : 8000) and atropin drops should be instilled. Iodoform salve (or powder) may be freely applied within the conjunctival sac; indeed, vaselin itself is efficient in these circumstances.

Internally, quinin, iron, and mercury have been recommended; but the greatest reliance should be placed upon diphtheria antitoxin, which should be promptly administered exactly as it is in ordinary faucial diphtheria. At the first dose 1500 to 2000 units of antitoxic serum may be injected into the lateral abdominal wall, and repeated in ten or twelve hours, according to the severity of the symptoms.

The sound eye may be covered with a bandage or Buller's shield. The patient should be isolated.

In addition to the deep-seated, necrotic variety of diphtheria of the conjunctiva, the disease, according to Uhthoff, Sourdille, Elschnig, and Morax, may assume a benign aspect and a superficial pseudomembranous form. Why virulent diphtheritic bacilli sometimes cause a superficial and sometimes a deep interstitial type of the affection has not, according to Uhthoff and Coppez, been determined. The former author also describes a simple catarrhal conjunctivitis in association with diphtheritic bacilli.

Phlyctenular Conjunctivitis (*Phlyctenular Ophthalmia; Scrofulous Ophthalmia; Eczema of the Conjunctiva*).—This is a form of inflammation of the conjunctiva, characterized by the appearance of one or more grayish elevations, situated chiefly upon its bulbar portion in the immediate vicinity of the cornea. Less frequently the phlyctenules appear upon the tarsal conjunctiva, those on the lower lid being of firmer consistence than those on the bulbar conjunctiva (Schiele).

Causes.—The disease is of constitutional origin, and its subjects are often tuberculous and badly nourished children. Errors of diet, unwholesome foods, and the abuse of tea and coffee act as predisposing causes. It often follows the exanthemata, especially measles. Infectious rhinitis is always present, and usually the submaxillary and cervical glands are swollen, and there is eczema of the lip and nares. There is a distinct clinical association between this disease and eczema. It is possible that the active micro-organism is the *Staphylococcus pyogenes aureus* or *albus*, which is found beneath the epithelium of the affected conjunctiva, but an endogenous origin of the disease cannot be wholly excluded. That the disease is a true tuberculosis is disputed by Axenfeld.

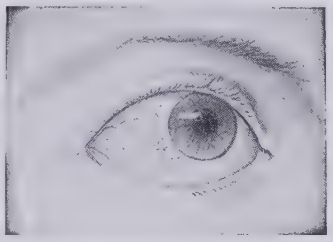


Fig. 109.—Phlyctenular conjunctivitis (Children's Hospital).

Symptoms.—The disease occurs in a single and a multiple form; the pimples or phlyctenulæ lie near the corneal margin or directly upon it, and are usually from 1 to 3 mm. in diameter.

If the elevations are large, yellow, and contain purulent material, the disease has been called *pustular ophthalmia*.

In any circumstances it is accompanied by dread of light, injected blood-vessels, and increased lacrimation. The conjunctiva may be transparent, or the disorder associated with a mucopurulent conjunctivitis. After the exanthemata this association is common.

In the multiple form, numerous minute phlyctenules may be scattered over the entire conjunctiva, and are accompanied by decided general red injection, irritation, and photophobia. The disorder subsides in from ten days to two weeks.

Treatment.—Locally, mild antiseptic collyria, especially lotions of boric acid, are useful. Much irritation indicates atropin drops and the *occasional* instillation of holocain to relieve the photophobia. The eyes may be protected by colored glasses.

After the acute symptoms have subsided the best results are obtained by introducing the yellow oxid of mercury, 1 grain to 1 dram (0.065–3.885 gm.), into the conjunctival sac or by dusting into it calomel, provided the patient is not taking iodid of potassium, otherwise a reaction between the potassium iodid in the tears and the calomel occurs, with the ultimate formation of double iodids, which are caustics (*calomel conjunctivitis*). Applications of borobismuth ointment to the nasal eczema are recommended by Schiele, who also speaks favorably of

such antiseptic powders as gallicin, iodogallicin, and bismuth oxyiodid tannate. Linear cauterization of the fornix has been advised in severe cases.

An excellent regulation treatment is a mild course of mercurial laxatives. Simple diet, good air, exercise, and internally, quinin, iron, arsenic, and cod-liver oil, complete the therapeutic measures. The nose and throat must be carefully examined and treated according to existing conditions.

Phlyctenular conjunctivitis is so closely allied to phlyctenular keratitis that the separation of the two affections is purely artificial, and this account is a preface to the description of the more exact disposition and relation of the phlyctenules, which appears on page 266.

Vernal Conjunctivitis (*Fruehjahr's Catarrh*, Saemisch; *Phlyctæna Pallida*, Hirschberg; *Periodic Hyperplastic Conjunctivitis*, Wicherkiewicz; *Fibroma of the Limbus; Spring Conjunctivitis*).—This form of conjunctival disease is characterized by photophobia, stinging pain, considerable mucous secretion, the formation of flat granulations in the palpebral conjunctiva, and a hypertrophy of the conjunctiva surrounding the limbus of the cornea.

Causes.—Definite information in regard to the cause of this disease is lacking. It is possible that some specific micro-organism exists which has not yet been isolated. Although frequently the disorder returns in the early spring, is more aggravated during the summer season and subsides in the fall and winter, cases may occur in any month of the year, and its designation, *spring catarrh*, is not a good one, because the affection is in no sense a catarrhal one, and it does not necessarily occur in the spring. Heat, especially dry heat, must be regarded as an exciting factor, even though it is not the sole cause of the disease. It is most frequent between the ages of five and fifteen, but occasionally occurs in advanced adult life and in very young children, even those but a few months of age. According to Posey's investigations, it is more frequent in males in the proportion of 85 to 15 per cent. and the greater liability of the male sex to this affection is confirmed by many observations. It may accompany the disease known as hay-fever. Some writers decline to consider vernal conjunctivitis a distinct disease, but look upon it as a hypertrophic form of chronic conjunctivitis. In a few instances it appears to be hereditary, and Meyerhof and Gabriélidès describe a familial type of vernal conjunctivitis. The latter author inclines to the belief that spring conjunctivitis may be a manifestation of an autointoxication of the organism. At times there appears to be a congenital disposition to the disease.

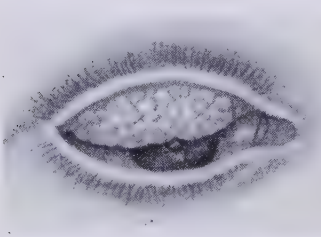


Fig. 110.—Vernal conjunctivitis (Haab).

Symptoms.—There are three varieties of this disease—the limbus,

palpebral, and mixed forms. The affection begins like an ordinary conjunctivitis and is almost always bilateral; a few unilateral cases are on record. There are photophobia, more or less mucous secretion, circumscribed pericorneal injection, and the formation at the limbus of small, gray, semitransparent nodules, which swell up and overlap the edge of the cornea. Occasionally in the limbus form of vernal conjunctivitis large vegetative lesions develop.

The conjunctiva of the bulb is injected, that of the lids is slightly thickened and of a dull, pale color, as if brushed over with a thin layer of milk. This milky film or "bluish-milky" surface reflex is one of the most characteristic symptoms of the disease, and may be observed before the granulations appear on the inner surface of the lids; occasionally, even in typical cases, it is lacking. When the granulations appear in typical form, they cover the tarsal conjunctiva, are flattened, and contain deep furrows between them. Sometimes they are very hard, large, and leathery. In the colored race there is a brownish pigmentation of the scleral base of the hypertrophied masses (Burnett).

The disease may be distinguished from trachoma by the flattened appearance of the granulations, the absence of infiltration and pannus, and the history of recurrence at special seasons of the year. Elschnig calls attention to an arrangement of the blood-vessels of the tarsal conjunctiva which he considers peculiar to spring catarrh. The normal vascular distribution is replaced by innumerable small vessels arising perpendicular to the conjunctival surface. Mixed forms of spring catarrh and trachoma have been described (May, Meyerhof).

The **pathologic histology** of the lesions has attracted much attention in recent years. According to Axenfeld, who has elaborately investigated this disease, the primary lesions arise in the subconjunctival tissue, followed by proliferation of the epithelium. An accumulation of plasma cells takes place, succeeded by a homogeneous sclerosis of the connective tissue. Elastic tissue is abundant. The milky appearance of the surface of the conjunctiva is due to subepithelial hyaline thickening. Eosinophils are frequent in the conjunctiva and occur in large numbers in the secretion. There is no essential difference between the tarsal and palpebral manifestations of the affection, unless future investigations should confirm the assertion of some observers that in the bulbar proliferations the epithelial changes take precedence.

The **prognosis** of the disorder is not unfavorable, except in so far as relapses are concerned, which make its course a long one, sometimes lasting from eight to ten years, or even longer. Usually the activity of the process begins to subside after it has existed for six to seven years, that is, the disease tends to be self-limited. Aggravated types of this affection of long duration, however, are well-known. Opacity of the cornea may result; indeed, slight changes, noted especially by slit-lamp examination, are not uncommon. Conical cornea may be a sequel.

Treatment.—During the height of the attack the eyes may be protected by dark or yellow-tinted glasses. Cold compresses afford some relief. Weak astringents and antiseptic lotions, such as have been

recommended for ordinary conjunctivitis, are useful. The application of boroglycerid to the everted lid is sometimes valuable, and the systematic use of a preparation of suprarenal extract, or of adrenalin chlorid (1 : 10,000), to which a 1 per cent. solution of holocain is added, is of service. Axenfeld suggests the application of a solution of sulphate of quinin, and Elschnig advises the instillation of a watery solution of ichthyol (1 to 2 per cent.). Salicylic acid ointment (1 per cent.) is recommended, and massage with yellow oxid ointment may be tried. Electrolysis has been employed, and brossage has been advised by L. W. Fox. Exuberant granulations and limbus hypertrophies may be excised. According to Wicherkiewicz, a collyrium of antipyrin (10 per cent.), and instillations of 2 or 3 per cent. of protargol are efficient. Fibrolysin, as suggested by Luedde, is a remedy of real value. It is used as follows: a 2 per cent. solution of holocain having been instilled, the lid is everted and painted with a $\frac{1}{5}$ per cent. solution of nitrate of silver, followed, after its neutralization, by an application of fibrolysin by means of a cotton-wound probe, or the fibrolysin may be dropped into the eye. These applications may be made every other day. In the intervals the patient should use a sulphate of zinc lotion ($\frac{1}{4}$ per cent.) freely. Starr, in Buffalo, and Allport, in Chicago, have recommended the *x*-rays in the same manner as they are applied in trachoma, and have reported favorable results. Radium treatment of vernal conjunctivitis is most effective. Shumway, who has well studied the influence of this agent in this respect and who has reported, among others, cases from the author's service in the University Hospital, advises the following technic: the eye being cocainized and the upper lid everted, the radium element (35–50 mg.) is applied to the exposed surface, enclosed in an aluminium tube 5 mm. in thickness. The application may be made at intervals of about four weeks. There is always some reaction; the lashes are apt to drop out, but in time are sometimes reproduced. There is some evidence to show that the internal administration of arsenic is of advantage. Associated intranasal inflammation should be treated. Change from a warm to a cool climate is of service.

Follicular Conjunctivitis (*Follicular Ophthalmia; Conjunctivitis Follicularis Simplex; Folliculosis; "School Follicles"*).—This affection is characterized by the presence of small pinkish prominences in the conjunctiva, for the most part in the retrotarsal folds, and usually arranged in parallel rows. The descriptive term "conjunctivitis" may be applied to the affection if the signs of inflammation are associated with it; if the latter are absent, the term *folliculosis of the conjunctiva* is more appropriate.

Causes.—The disease arises under the influence of poor hygienic surroundings, especially in pauper schools, where it may appear as an aggravated epidemic, but it is frequently seen in comparatively mild form especially among children during their school years, particularly if they are the subjects of anemia and chlorosis; adenoids and granular pharyngitis are commonly present. Indeed, in so large a percentage of school children can tumefaction of the conjunctival lymph-follicles be

found that the name *school-folliculosis* has been suggested by Greeff. Evidently refractive errors are an exciting cause of many of these cases. Enlargement of the follicles may also be caused by local irritants and some medicaments—for example, atropin (see page 250).

Much difference of opinion exists as to whether folliculosis should be placed in a separate category from trachoma, or whether it should be regarded as an early stage of the latter disease. Although transitional forms apparently exist, the evidence, clinically at least, warrants the belief that this affection is distinct from trachoma, because folliculosis occurs where trachoma is unknown, and because the follicles disappear without leaving a trace of their existence or producing lesions in the conjunctiva. Histologically, however, there is no decisive difference between fresh follicles and fresh trachoma bodies. It would seem, as Greeff insists, that folliculosis may arise under the influence of various excitants, and in this sense is a symptom and not a distinct disease.

Symptoms.—The children—for it mostly occurs in children and young people—complain of slight dread of light and inability to continue at close work, and inspection reveals numerous round elevations in the conjunctiva, chiefly along the fornix, which are tumefied lymphatic follicles, that is, they represent an enlargement or elaboration of the normal lymphoid follicles, due to an irritant, perhaps chemic in nature. The color of the follicles varies from nearly white to a decided pink. After their disappearance the conjunctiva regains its natural state.

If with the enlarged follicles inflammatory symptoms are combined, the disease is a true *follicular conjunctivitis;* the lids are swollen, reddened, and their margins streaked with secretion, which, at first thin, may become more purulent and quite abundant. In certain circumstances the disease resembles the condition termed "swelling with catarrh" (see page 213), except that the development of the follicles is much more evident. The inflammatory form of the affection may assume a more *chronic type,* with special development of the follicles in the fornices, and only secretion enough to stick together the lids in the morning. Often an acute mucopurulent conjunctivitis precedes the development of the follicles, that is, they are evident after its subsidence.

Diagnosis.—The disorder can usually be distinguished from trachoma by observing that the small bodies, which are benign follicles, are neither so large as trachoma granulations nor so highly colored as hypertrophied papillæ; that they are confined to the fornices and are not seen on the plica or bulbar conjunctiva; that the mucous membrane is not affected more deeply than the lymphatic follicles; and that cicatricial changes are not present. Borderline cases, however, occur, which are difficult to classify and of which no one has ever yet succeeded in writing a description upon which an entirely satisfactory diagnosis could be made.

Prognosis.—This is good in so far as the fate of the mucous membrane is concerned, but the disorder is troublesome and often lasts for

months, and under imperfect hygienic surroundings and in crowded asylums may prove to be a stubborn affection.

Treatment.—The usual antiseptic and astringent lotions are indicated and applications of boroglycerid or of tannin and glycerin are useful. A salve of $\frac{1}{2}$ grain (0.0324 gm.) of sulphate of copper to the dram (3.885 gm.) of vaselin has been highly extolled. If there is much secretion, the usual treatment of conjunctivitis is required, especially silver and its various salts.

Refractive error should be corrected because ametropia aggravates the disorder. In stubborn cases, and in those where the follicular eruption is elaborate, especially in asylums and schools, expression of the swollen follicles with suitable forceps should be performed (see page 718).

Trachomatous Conjunctivitis (*Trachoma; Granular Lids; Granular Conjunctivitis; Egyptian Ophthalmia; Military Ophthalmia*).—This is a disease of the conjunctiva in which this membrane loses its smooth surface owing to an inflammatory infiltration of its adenoid layer, associated with the development of follicles ("granulations") and enlargement of the so-called papillary layer. After absorption and metamorphosis of the inflammatory material, cicatricial changes are found.

Causes and Distribution.—Formerly it was the custom to separate this disease into two forms—*acute granulations*, or *acute granular conjunctivitis*, and *chronic granulations*, or *chronic granular conjunctivitis*, and certain systematic writers—for example, Saemisch—continue this distinction. The author is in agreement with those who maintain that the so-called acute trachoma, at least in the majority of cases, represents an admixture of ordinary trachoma and acute conjunctival catarrah. An attack of acute conjunctivitis may precede the development of trachoma. According to MacCallan, in Egypt, the acute symptoms are most often caused by the Morax-Axenfeld bacillus, the Koch-Weeks bacillus, and the gonococcus. Greeff, however, seems to have proved by inoculation that there is an acute trachoma which may develop in a few days.

Trachoma may arise apparently under the influence of poor hygienic surroundings, and in institutions where the inmates are crowded together the disease may readily spread.

Distribution.—The dissemination of trachoma in Europe became noteworthy after the return of Napoleon's soldiers from Egypt, inasmuch as 75 per cent. of them had been infected. They came repeatedly in contact with each other and with the civil population, and thus spread the disease, often in epidemic virulence.

Hence in the early portion of the nineteenth century trachoma found in civil life a favorable soil for its dissemination in jails, asylums, and wherever inhabitants of the poorer classes dwelt together in close contact, and at this time in many pauper schools every inmate was affected. The acute course which the disease manifested at this period of its history was doubtless due to mixed infection, and such epidemics have in great measure disappeared. Nevertheless, trachoma remains

endemic in many lands; it is, indeed, a world disease. It is most frequent in Arabia and Palestine, and in Egypt it is generalized, fully 95 per cent. of the native population being affected (MacCallan). Trachoma is endemic in Syria, Persia, Central Asia, China, and Japan, and is exceedingly prevalent in Eastern Europe, especially in Gallipoli, Poland, Lithuania, Russia, Hungary, and certain districts in Prussia; it is noteworthy that Jews of inferior social grade are prone to be affected. It is frequent among Italians of the lower orders, especially in the south of Italy. In England "trachoma is an alien disease, imported by aliens, propagated by aliens, and handed on to the native population by aliens" (Parsons); in Ireland it is common among the poorer classes.

The menace of trachoma on our own shores is one of the serious problems of our immigration officers. It is common among native Americans in certain portions of our country, moreover, in severe and destructive manifestations, especially in definite areas in Illinois, in the mountainous regions of Kentucky and West Virginia, and is particularly noteworthy, according to the researches of Dr. Stucky and of Dr. John McMullen, of the United States Public Health Service, in the neighborhood of the junction of Kentucky, Tennessee, and the Virginias. In States where high winds prevail and there is much irritating alkaline dust, *e. g.*, in Oklahoma, Arkansas, Texas, Arizona, and New Mexico, trachoma is very prevalent. To the prevalence of trachoma among the Indians of our country much attention has been paid, with encouraging results.

A certain racial predisposition to trachoma has been maintained, the Mongolian race being especially susceptible; but throughout Asia the disease is no respecter of race, the Aryan, Semitic, and Mongolian suffering with equal frequency. Although the negro is far less liable to trachoma than white people, he is not exempt, as has been demonstrated by Minor and White, and the author has observed a number of cases in a large experience in the Philadelphia General Hospital, although he cannot be sure that these negroes were of pure blood.

A climatic predisposition is more than doubtful, although it has been found that dwellers in certain regions of the earth where the climate is damp are readily affected. While it has been maintained that an altitude of more than a thousand feet confers a comparative immunity from the disease and facilitates its cure, and while this may be true in Switzerland and the Tyrol, certainly in our own country no such influence of altitude is evident. D. W. White has found and studied the disease 8000 feet above sea level.

Bacteriology of Trachoma.—Transference of the morbid material from a trachomatous conjunctiva to another eye may result not only in a purulent conjunctivitis, but in a disease like the one from which it came. In this sense the disease is specifically communicable, although the affection is not conspicuously communicable by secretion-inoculation (Axenfeld). There is no proof that trachoma is caused by microorganisms of the bacterial group or by blastomycetes. Treacher Collins has suggested that the disease depends upon an organism of ultrami-

croscopic dimensions. Cohen and Noguchi think the cause of trachoma is a non-identified specific virus.

Halberstädter and von Prowazek, working in Java, and Greeff, Frosch, and Clausen, in Germany, discovered in the discharge and follicle contents of trachoma very small granules, resembling diplobacteria. They are surrounded by a zone (hence called by Prowazek *chlamydozoa*) and occur either isolated or grouped together within the cell next to the nucleus (see Figure 111). They increase in number, and gradually occupy almost the entire protoplasm of the cell which is destroyed, and they are set free in the secretion. Certain small bodies are seen in the protoplasm of the cells and outside of the cells, and they almost always accompany the "inclusions," and are called "Lindner's initial bodies." The Prowazek bodies are found in fresh, untreated trachoma, less easily in granular conjunctivitis of long standing. Their exact nature is unknown, although, according to Axenfeld, they probably are neither cell-products, metamorphosed

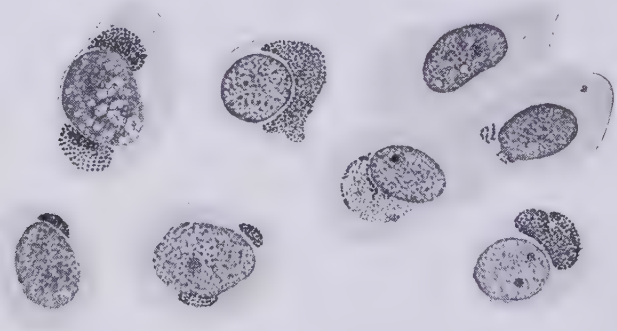


Fig. 111.—So-called trachoma bodies—epithelial inclusions (Axenfeld).

gonococci (Herzog), nor the products of mucoid degeneration. They are so seldom present in other conjunctival disease that their detection is significant. Their absence, however, does not exclude trachoma; in other words, they are not pathognomonic. They are usually known as "Prowazek or Prowazek-Halberstädter bodies or corpuscles," and they have also been found in ophthalmia neonatorum (Heymann) (see page 215), in their early stage in the normal conjunctiva, and in some forms of chronic conjunctivitis (Erdmann).

Pathology and Varieties of Trachoma.—The pathognomonic appearance and essential element of the disease trachoma are the "granulations," or "trachoma bodies," or follicles.[1]

Two views have been held—the one that the trachoma bodies have a special pathologic character; the other that they are derived from

[1] It should be remembered that the word "granulations" refers to the characteristic feature of trachoma, and not to surface granulations which may form during the course of the disease. The Prowazek bodies are also called "trachoma bodies" by some authors, for example, by Greeff.

the lymphatic follicles, which, although poorly developed, are probably present in the natural human conjunctiva, and some authors declare that these follicles and their changes originate all the anatomic and

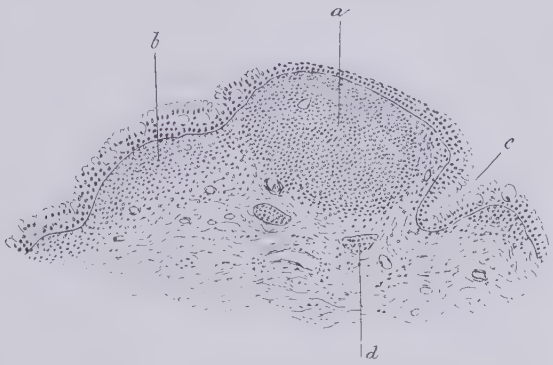


FIG. 112.—Trachoma of the retrotarsal fold: *a*, Follicle; *b*, diffuse infiltration; *c*, Henle's gland with goblet-cells; *d*, lymph-vessel filled with leukocytes (× 30). (Holden.)

clinical qualities of trachoma. Although it may not be possible to distinguish in the early stages trachoma bodies from enlarged lymphatic follicles, there is a difference in the nature of the two conditions, and for the most part the "unitarian standpoint" with reference to trachoma has been abandoned —that is, the theory that all follicles in the conjunctiva represent trachoma. Certain so-called "benign follicles" exist which disappear without a residue of lesions (see page 234), and, on the other hand, an infection associated with follicular formation arises which subsides after a long period of time and leaves cicatrices (see page 240). Between these two forms are the "borderline" cases which are difficult to classify. Treacher Collins suggests that in trachoma there is an invasion of the subepithelial tissue by microorganisms which have penetrated the outer defence of epithelium and have become surrounded by a new formation of lymphoid tissue. The onset of trachoma may be preceded by a Koch-Weeks bacillus conjunctivitis in which Prowazek bodies can be found. Some



FIG. 113.—Chronic trachoma of the papillary type, beginning cicatrization (Medical War Manual, No. 3).

authors deny that trachoma is a specific disease, believing that it is the pathologic expression of the reaction of the conjunctiva to various irritants (Walker).

The following clinical varieties of chronic trachoma have been recognized by systematic writers:

1. *Papillary trachoma,* in which the trachoma bodies or follicles are sparsely present or are hidden from view by hypertrophied conjunctival papillæ, or, more accurately, pseudopapillæ. The blood-vessels are enlarged, and there is marked increase in the number of lymphoid cells. The follicles in the adenoid layer lift above them the thickened epithelium. This form is sometimes called *chronic trachoma* (Fig. 113).

2. *Follicular trachoma,* in which the presence of the "follicles" or trachoma bodies is the chief characteristic. These bodies are collections



Fig. 114.—Follicular trachoma (Medical War Manual No. 3).

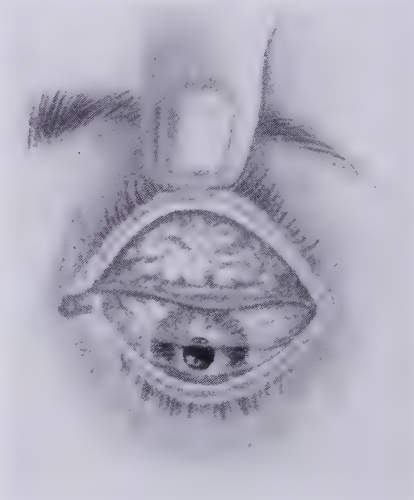


Fig. 115.—Cicatrical trachoma and pannus (Medical War Manual No. 3).

of round lymphoid cells which may possess an incomplete capsule, and which, as before stated, are elaborately developed in the adenoid layer of the conjunctiva. Some authors consider follicular conjunctivitis (see page 234) a variety of this type. Systematic writers have differentiated the following cellular elements in the trachoma follicle: lymphocytes, which form the chief constituent of the peripheral zone; mononuclear leukocytes, which compose the chief portion of the follicle; phagocytes, which are found among the leukocytes; and certain accessory elements—for example, multinuclear cells, etc. Beneath the follicles are dilated lymph-vessels, and blood-vessels may extend into the follicles. The lymphadenoid tissue surrounding the follicles is infiltrated with leukocytes. Some of the cells of the follicles are discharged or absorbed; others are converted into connective-tissue fibers, which, by their contraction, produce the changes described on page 241.

According to Parsons, the invariable termination of trachoma in cicatrization is brought about by absorption of the contents of the follicles and proliferation of the connective tissue of the conjunctiva, it being doubtful if the elements of the follicle can themselves form fibrous tissue.

In one form, designated by H. Knapp *non-inflammatory follicular trachoma*, the spawn-like granulations develop in the conjunctiva without evidence of inflammation, and have been regarded as analogous to nasopharyngeal adenoid hypertrophies (see also page 234).

3. *Mixed trachoma*, in which the follicles or bodies lie among hypertrophied and inflamed papillæ, but are not hidden by them. This type is sometimes described as *diffuse* or *complicated trachoma*.

4. *Sclerosing trachoma*, in which, after an initial stage of ordinary granulations, leathery (fibrous), flattened excrescences develop in the upper tarsal and retrotarsal conjunctiva.

5. *Cicatricial trachoma*, in which atrophy and scar tissue are manifest—"the end stage of uncured cases" (H. Knapp).

Although the separation of trachoma into these varieties may be convenient from the clinical standpoint, such a separation cannot be maintained on histologic grounds. Indeed, Saemisch maintains that the terms "papillary" and "follicular trachoma" should be avoided, as the first corresponds with blennorrhea, and the second with follicular conjunctivitis. Certainly the term "follicular trachoma" has given rise to a good deal of confusion in its various interpretations and it would be well if it were eliminated by universal consent.

Symptoms.—The "granulations or follicles" often appear without antecedent inflammation, and so insidiously that their real nature is for a time unknown to the patient. They usually arise in the form of grayish-white, semitransparent bodies, which vary in size according to their stage of development, and which, from fancied resemblances, have been called "sago-grain" or "vesicular" granulations. They may be disseminated or arranged in parallel rows, and have sometimes been likened to the appearance of frog's spawn (*follicular trachoma*). The granulations are, for the most part, confined to the palpebral conjunctiva, and the upper retrotarsal fold, which is a favorite location, should be well exposed during the examination. They are also found on the bulbar conjunctiva, the caruncle, and semilunar folds.

The mucous membrane is pale or yellowish red, unevenly rough, and contains the trachoma bodies, or follicles, which have a more or less deep situation and fill up the tissue. If they have not followed an acute process, there are few or no irritative manifestations and little discharge —perhaps only sufficient to glue together the lids. As time goes on the closely packed masses compress the true conjunctival tissue and its circulation, and a superficial vascularity of the cornea may appear. This stage may last for months and be subject to numerous variations.

In the next stage vascularity is increased, the follicles grow larger, soften, and their contents are forced out by the pressure of the surrounding infiltrations, forming, in association with the hypertrophied

conjunctival papillæ, red protuberances (hypertrophied pseudopapillæ). This period is associated with strong irritation and mucopurulent or purulent secretion, photophobia, local pain, and corneal complications.

During the time of fatty degeneration and softening, which by some authorities is deemed a process of ulceration, fresh follicular (granular) eruptions take place, in turn to go through the same changes which their forerunners have undergone. The mucous membrane now has a flesh-red appearance; it is with difficulty that the "granulations" are distinguished from the papillæ, and, indeed, they are united with them, forming variously shaped diffuse or isolated protuberances. Sometimes resorption of the follicles, which can take place at any stage, appears to occur by retrogression without softening. Usually degenerative changes in the cells take place. Fusion and softening of the closely packed follicles may be followed by hyaline degeneration, giving rise to a gelatinous appearance, which is sometimes designated *gelatinous trachoma*.

In the final stage cicatrization begins, and grayish-white scar-lines appear, intersecting the remains of the old "granulations." If these cicatrices lie parallel to the ciliary borders, they present, on eversion of the lid, a typical appearance[1] (Fig. 115).

By a gradual process of cicatrization of the old "granulations" and by the advent of successive new crops, a chronic induration and diffuse scar tissue results (*cicatricial trachoma*). This being firmly attached to the tarsus, which itself has undergone inflammatory and lymphoid infiltration, contracts, and the deformities of the lid and its border, so common in this disease, result. The fibroid induration of the mucous membrane affects all portions, and there may be almost entire obliteration of the conjunctival sulcus, or the membrane may undergo a species of drying up, to which the name *xerosis* has been applied. Individuals with granular conjunctivitis, in the stage of thickening of the mucous membrane, have an almost characteristic sleepy look, peering uncertainly through narrowed palpebral fissures, caused by the ptosis-like droop of their indurated eyelids.

In so-called *acute granular conjunctivitis* the lids are swollen, the conjunctiva reddened, the conjunctival pseudopapillæ hypertrophied and elevated by the underlying lymphoid infiltration, while between them are found the yellowish, round "granulations." The dread of light is intense, and on forcible separation of the lids scalding tears gush out, and later mucopurulent discharge appears. Still later, vascularization and ulceration of the cornea may develop. Such manifestations are not a special form of trachoma, but should be regarded as a mixture of granular conjunctivitis and catarrh; acute exacerbations of chronic trachoma are common (see also page 236).

Sequelæ and Complications of Trachoma.—The most important results of long-standing trachoma are trichiasis, distichiasis, and entropion, conditions already described (see page 197), atrophy and shrink-

[1] It is convenient to divide the disease into three stages, as Raehlmann has done, but it is not always possible to separate sharply each stage by symptoms or appearances peculiar to itself.

ing of the conjunctiva from cicatricial changes (see page 254), cloudiness and ulceration of the cornea, and *pannus*.

Pannus is the development on the cornea of a gelatinous vascular tissue, which usually begins in that portion of the cornea covered by the upper lid (sometimes below or at one side), but which, in severe cases, may involve its entire surface. It depends upon the formation of new blood-vessels between the corneal epithelium and Bowman's membrane, associated with collections of round cells. It may be composed of only a few vessels (*pannus tenuis*), or be thick, fleshy (*pannus crassus*), and bulging in appearance. If softening and *ulceration* occur, the true corneal tissue is invaded.

Pannus is not a simple traumatic irritation due to the action of the roughened lid (although this may be a predisposing factor), but is a special implantation of the trachoma process on the layers of the cornea. Extensive and deep ulceration may complicate pannus, which, in turn, may lead to the development of iritis; or the cornea may become entirely opaque; or, finally, the ulceration may be followed by perforation of this membrane and staphylomatous bulging. The *ulcers* may begin at the border of the pannus (a favorite situation) or within the area of the pannus, or in some portion of the cornea not otherwise affected.



FIG. 116.—Typical pannus with line of demarkation (Medical War Manual No. 3).

In some of the subjects of trachoma dacryocystitis is present and trachomatous changes may be detected in the walls of the sac and even in the nose. The inner end of the canaliculus is frequently occluded in trachomatous patients. Although trachoma of the lacrimal sac is well recognized, MacCallan doubts if *trachomatous dacryocystitis* is ever a primary condition. Reinfection of the conjunctiva from a trachomatous lacrimal sac has been observed.

Diagnosis.—An examination of fresh material should reveal the Prowazek-Greeff granules, but, as has already been pointed out, the presence of these bodies, while significant, is not sufficient to establish a diagnosis, *i. e.*, their absence does not exclude trachoma. The disease is made evident by direct inspection of the everted lids unless the associated swelling of the papillæ is so great as to obscure the "granula-

tions," especially in the forms of so-called papillary trachoma. Hypertrophied conjunctival papillæ, chronic blennorrhea, and surface granulations must not be mistaken for trachoma; the thickening and induration of the tarsus is a distinguishing feature in granular conjunctivitis. The clinical distinctions existing between trachoma and follicular conjunctivitis have been pointed out (see page 235). The distinctions between vernal conjunctivitis, Parinaud's conjunctivitis, and tuberculosis of the conjunctiva are elsewhere described. In early stages of trachoma loupe-investigation of the upper portion of the cornea will not infrequently detect a delicate ingrowth of vessels (early stage of pannus) not discoverable with the naked eye (Stieren and Van Kirk). Great care should be exercised in the inspection of immigrants, and those undoubtedly trachomatous should be deported. All suspected persons, and all those in whom the diagnosis is uncertain, should be isolated and detained until the exact nature of their conjunctival trouble is ascertained.

Prognosis.—In the best circumstances, trachoma, when well established, is a tedious disease, and greatly endangers the vision of the patient. Relapses are frequent, and at any time the disorder is likely to assume an intense inflammatory action. Its communicable character renders the affection especially dangerous in schools and in any institution where large numbers of inmates are gathered together. The discharge, even when present in slight degree, is readily conveyed from one subject to another by the careless use of towels and common utensils. Great caution is necessary in such circumstances to prevent a disastrous epidemic. Trachoma, if properly managed, is curable and improvement in prognosis in recent years, under the influence of well considered operative procedures and medicamental applications, has been evident.

Treatment.—The treatment of chronic trachoma includes the application of caustics, astringents, antiseptics, and certain so-called specific remedies, operative procedures, and general medication.

Local applications of astringent and caustic preparations are used to cause absorption of the "granulations," but these should not be of such strength as to produce cicatricial changes more harmful than the original malady.

A variety of substances has been employed; indeed, it is safe to assert that there is scarcely an antiseptic or caustic agent the use of which is permissible in ocular disorders that has not been tried in the effort to alleviate the symptoms of this disease. If granular conjunctivitis is associated with much discharge in the sense of mucopurulent secretion, the ordinary antiseptic and slightly astringent lotions are useful, and should be freely employed to irrigate the conjunctival culdesac. Those which serve the best purpose are saturated solutions of boric acid, bichlorid of mercury (1 : 5000 or 1 : 10,000), and cyanid of mercury (1 : 2000), and mercurophen (1 : 8000). Sulphate of zinc (0.5–1 per cent.) has been much employed and was found of distinct advantage in the routine treatment of trachoma during the late war.

Formerly, during the stage of conspicuous lymphoid infiltration and decided follicular eruption, without the presence of much discharge, the direct application to the everted lids of strong solutions of bichlorid of mercury (1 : 300 or 1 : 500) were much employed (see also page 720).

In the stage of softening of the granulations and swelling of the conjunctival pseudopapillæ, associated with mucopurulent and purulent discharge, in addition to flushings with the antiseptic solutions already mentioned, nitrate of silver is of value, employed in the manner already described (see page 221). Generally it is not necessary to use a solution stronger than 2 per cent. Instead of nitrate of silver, argyrol and protargol in the usual strengths may be employed. In this stage solutions of permanganate of potassium (1 : 3000 and 1 : 5000) have been advocated, while stronger solutions (1 : 1000) have been applied directly to the everted lid.

At one time sulphate of copper was almost universally employed in the treatment of trachoma, and the author is convinced that it still occupies a most useful place in the management of this affection. The value of sulphate of copper depends upon its power to excite phagocytic activity. Where the eruption of new granulations is associated with beginning cicatricial metamorphoses of old crops and their surrounding tissue, this remedy is of advantage. The crystal of sulphate of copper should be smooth and carefully applied to all portions of the affected areas, especially to the retrotarsal folds, and the treatment followed by washing the surface with cold water. It is a painful remedy, and in sensitive patients there is no objection to holocainizing the eye. In order to render sulphate of copper painless, it has been suggested to fuse it into a crayon composed of this drug, orthoform, holocain, and gum tragacanth. Sulphate of copper dissolved in glycerin (5 per cent. solution) is a most useful application.

In place of sulphate of copper, copper citrate (cuprocitrol), originally recommended by F. R. von Arlt, has found favor with some surgeons. It may be employed in a 5 or 10 per cent. ointment, which is introduced well in the conjunctival sac, and gentle but thorough massage used immediately afterward.

During the later stages of trachoma, in order to hasten the absorption of remaining granulations and perhaps to prevent the tendency to xerosis, boroglycerid (30–50 per cent.) is a useful remedy, applied in the usual manner with a cotton mop. In mild cases, or after an impression has been made with stronger caustics, a favorite astringent is tannin and glycerin, 30 to 60 grains to 1 ounce (1.95–3.9 gm. to 30 c.c.), or the everted lids may be touched with an alum crystal. Among the many additional remedies which have been tried in this affection the following may be mentioned: Liquid carbolic acid, liquor potassa, betanaphthol, hydrastin, iodoform, or aristol (in powder or salve), an ointment of the yellow oxid of mercury, calomel, iodid of silver, ichthargan (2 to 3 per cent.), itrol in powder, ichthyol, and cyanid of mercury (1 : 500), which is energetically rubbed by means of a tampon of cotton wool over the granular surface.

Trachoma is liable at any time to develop acute symptoms: increased discharge; exacerbation of pannus, with clouding and ulceration of the cornea; hyperemia of the iris, and acute pain in the brow and temple. Usually severe local applications must be discontinued, and the treatment instituted which is applicable to acute conjunctivitis, and which need not be here repeated. Hot compresses are often agreeable, and the pupils should be dilated with a solution of atropin or scopolamin. In this stage and, indeed, in other stages, especially if pannus is present, it would seem that dionin is of some value.

The *x-ray treatment of trachoma* has occupied a large share of attention within the last few years, and has been recommended by Mayou, Stephenson, Walsh, and other surgeons.

The author's experience with this method of treatment is too limited to render an expression of opinion from him of value. In the few cases in which he has used it and seen it employed the results were indifferent, certainly not any better than those obtained by ordinary therapeutic agents or operative procedure.

Radium has also been used in the treatment of trachoma, but Charles H. May, who has studied the action of this substance in this respect, concludes that the results obtained are not so favorable as those secured with sulphate of copper. Carbon dioxid snow has also been employed; applications are made for a few seconds once a week at first, later the time may be increased to twenty seconds. It is commended by Treacher Collins and Tyrell.

Stephenson and Walsh also recommend the application of the *high-frequency current* through a vulcanite electrode applied to the upper lid in the treatment of severe trachoma.

Operative Procedures.—These include the various methods for removing the granulations: Scarification of the conjunctiva; abscission of the granulation; excision of the retrotarsal fold or of a strip of the infiltrated fornix; removal of a part of the tarsal conjunctiva at the same time that the strip of infiltrated fornix is excised (the so-called combined excision); extirpation of the tarsus (Kuhnt's extirpation) and squeezing or rolling out the trachoma follicles with suitable instruments, especially with Noyes', Knapp's, or Kuhnt's forceps. Removal of the granulations by means of a curet or stiff brush, and then rubbing into them strong solutions of bichlorid of mercury or cyanid of mercury (*grattage, brossage*), are measures that have been much employed. Grattage is performed by D. H. Coover with strips of sterilized sandpaper. The methods of applying the various operative procedures are described on page 718.

Of the methods just enumerated, expression of the follicles with suitable forceps, particularly in the so-called follicular forms of trachoma, is the most satisfactory, although both simple and combined excision of the infiltrated fornix sometimes yields exceedingly satisfactory results. Some surgeons, notably Mr. George Lindsay Johnson, recommend electrolysis in the treatment of trachoma.

Treatment of Trachoma Complicated with Pannus.—If the pannus is

limited in degree, it requires no special treatment, as it will disappear with the absorption of the granulations; but if it is extensive, and especially if associated with ulceration, special treatment should be directed toward its cure. This includes the local remedies which are appropriate for a vascular keratitis, namely, an antiseptic lotion, the various mydriatics, and occasionally dionin.

Inveterate pannus, without ulceration of the cornea, at one time was treated by the production of a violent conjunctivitis, characterized by the formation of a somewhat clinging false membrane, with a 3 per cent. infusion of *jequirity*, painted upon the everted lids. This method was introduced by de Wecker to substitute the old-fashioned inoculation of the conjunctiva with blennorrheic pus, and very properly appears to have been abandoned.

Since the introduction into ophthalmic practice of *jequiritol* and *jequiritol serum* by Roemer, these substances have been much employed in the treatment of trachoma. Jequiritol is an extract made from the seed of the *abrus precatorius*. It is used in a sterile solution mixed with 50 per cent. glycerin, so that an exact dose can be given without evil effects, which was not possible with abrin or the old infusion. According to Hoor, jequiritol is indicated in old trachomatous pannus with cicatrized and degenerated conjunctiva. It is contraindicated in purulent processes of the cornea, in recent opacities, and in fresh trachomatous pannus. In spite of all care certain complications may arise—for example, edema of the lids, pain, facial eczema, and suppuration of the lacrimal sac. Evidently jequiritol is not without danger, and should be restricted if used at all to the cases already described.

The operation of *peritomy*, which consists of an excision of a ring of conjunctival tissue surrounding the cornea, has been much practised for the relief of severe pannus. Another method is to scrape away the opaque and vascular areas in the cornea with a small knife (Gruening). If the palpebral fissure becomes contracted by cicatricial changes, or if during inflammatory periods in trachoma the lids dangerously compress the cornea, the operation of *canthoplasty* affords relief.

General Medication.—It is a mistake to depend solely upon local measures for the relief of granular conjunctivitis, for, although the disease has no proved constitutional origin, its subjects give frequent evidence of malnutrition, and are sometimes affected with tuberculosis. Hygienic surroundings, iron, cod-liver oil, hypophosphite of lime, arsenic, and, in short, a general tonic regimen are indicated. Suitable attention to the alimentary tract is important. The internal administration of iodid of potassium has been advised (Brown Pusey).

Parinaud's Conjunctivitis (*Infectious Conjunctivitis; Septic Conjunctivitis; Lymphoma of the Conjunctiva* [Goldzieher]; *Leptothricosis conjunctivæ* [Verhoeff]).—This rather rare form of conjunctival affection was first accurately described by Parinaud in 1889, and has in recent times been the subject of very extended researches, particularly by Chaillous in France, and Gifford, Verhoeff, and G. S. Derby in this country. According to the last-named authors, the disease has been

observed only in the temperate zone, and occurs a little more frequently in the autumn than at other seasons. The sexes are about equally affected, and all ages seem liable to it, the youngest patient recorded being one and a half years of age and the oldest fifty-nine. More commonly it is a unilateral than a bilateral disease; indeed, in only a very few instances have both eyes been affected.

The chief symptoms are the following: Swelling of the lid, usually most marked in the upper lid, hyperemia and edema of the bulbar conjunctiva, and a moderate mucopurulent discharge. The characteristic conjunctival lesions consist of large, reddish, semitransparent polypoid vegetations, small yellowish granules, erosions, and superficial ulcers. Sometimes the conjunctival growths are pedunculated. Very rarely corneal changes in the form of keratitis have been described. Glandular involvement usually takes place simultaneously with or very soon after the development of the ocular disease. In a few instances it has preceded them. Most often the preauricular glands are affected; more rarely the retromaxillary, the parotid, the submaxillary, and the cervical. Occasionally acute tonsillitis has been noted. The disease may last from one to five months.

So far, investigations have failed to isolate any of the known microorganisms as a causative agent of this disease, although McCrae found in one case a bacillus which resembled the Klebs-Löffler bacillus, which he regarded as the probable excitant of the ocular inflammation. Sinclair and Shennan isolated two varieties of white staphylococci from the necrotic areas in one case. Parinaud believed that the disease was of animal origin, and Hoor maintains that in the majority of cases there is a history of possible animal contagion; but Verhoeff and Derby regard this theory, at present at least, as unsubstantiated.

Herrenschwand has investigated a form of conjunctivitis similar to Parinaud's attributed to the *Bacillus pseudo-tuberculosis rodentium*, an observation which he thinks lends support to the theory that Parinaud's conjunctivitis is of animal origin. Recently Verhoeff has found in the lesions of this a section of filamentous organism classified as a *leptothrix*, which he regards as the cause of the disease. There may be difficulty in distinguishing the disease from tuberculosis of the conjunctiva (which is said to be associated with the lesions in some cases), and to reach a definite diagnosis it may be necessary to inoculate the anterior chamber of a rabbit's eye with a fragment of the suspected tissue. Microscopic examination of the excised tissue reveals cellular infiltration, consisting of lymphoid and phagocytic cells and marked cellular necrosis. According to Verhoeff the essential lesion is a focal area 3 mm. in diameter or larger, backed with endothelial phagocytes loaded with broken-down chromatin granules, which is situated just beneath the epithelium.

The **treatment** recommended includes the ordinary antiseptic collyria, nitrate of silver, or the newer silver salts, applications of sulphate of copper, and excision of the granulations. The injection of antidiphtheritic serum has been tried, and Sinclair and Shennan have instituted

vaccine treatment, but the patient did not remain under observation for a sufficient time to demonstrate the value of the method.

Sporotrichosis of the conjunctiva has been reported by Morax, Cruchandeau, Gifford, A. Knapp, and others. Thickening of the lid, nodular swelling of the conjunctiva, superficial yellowish ulcers, and adenopathy were present. (See also page 182.)

Chronic conjunctivitis (*chronic ophthalmia*), the result of an acute blennorrhea, has been referred to on page 226.

As an independent disorder, and assuming more the type of a hyperemia, it is a common disease in elderly persons. There are hyperemia, thickening of the papillary layer of the tarsal conjunctiva, swelling of the caruncle, soreness of the edges of the lids, and slight mucopurulent discharge. Often the bulbar conjunctiva is notably injected. Chronic conjunctivitis due to hypersecretion of the Meibomian glands (*conjunctivitis meibomiana*) and to "insufficiency of the eyelids," so that they close only with effort and remain open during sleep, is described by Elschnig. The latter may result in a form of xerosis of the conjunctiva (*tyloma conjunctivæ*, Saemisch). Long-standing conjunctivitis is occasionally due to molluscum contagiosum—the so-called *molluscum conjunctivitis* (H. and S. Gifford). A chronic conjunctivitis of moderate severity with only slight injection and traces of abnormal secretion in the commissural angles has been called *conjunctivitis sicca*, and is especially aggravating on awakening. It is to this affection that the so-called "morning ptosis" is usually due, the patient being unable to open the eyes except with the help of the fingers, which elevate the lid. The relation of the diplobacillus to chronic and subacute conjunctivitis has been described (see page 211; see also Hyperemia of the Conjunctiva, page 205). In association with chronic conjunctivitis and chronic ciliary blepharitis delicate *flame-shaped marginal keratitis* may arise, the lesions having their bases at the limbus and their apices advanced about one-third way across the cornea (W. T. Holmes Spicer).

Treatment.—Cleanliness, with antiseptic lotions, the application of "lapis divinus," an alum crystal, or glycerol of tannin, gr. x to f℥j (0.65 gm. to 30 c.c.), are useful local measures. Aqueous solutions of suprarenal extract (8 per cent.) or adrenalin chlorid (1 : 10,000) will temporarily dissipate the congestion, but they are not curative in their action. The puncta lacrimalia should be examined, and if they are closed, they should be dilated and the lacrimal passages irrigated with an Anel syringe, and the nasal chambers and paranasal sinuses should be carefully treated. Refractive error, which may keep up congestion, requires correction. For conjunctivitis meibomiana emptying of the Meibomian glands is necessary (Elschnig, Fridenberg). Boric acid in lanolin (2 per cent.) is useful if the conjunctival surface is too dry. If the Morax-Axenfeld bacillus is present, solutions of zinc sulphate or chlorid should be used (see page 213).

Egyptian and **military conjunctivitis** are terms which have at different times been loosely used to describe all forms of conjunctival

inflammations occurring in crowded barracks and similar institutions, which assumed an epidemic tendency, pursued a more or less chronic course, and hence included varieties of acute and chronic blennorrhea and mucopurulent conjunctivitis, in addition to those cases which possessed as a fundamental diagnostic symptom "granulations" of the conjunctiva, and which eventuated in the formation of cicatrices.

Lacrimal conjunctivitis is really a form of chronic conjunctivitis depending upon obstruction of the lacrimal passages and the frequently associated blepharitis, and in the discharge of which *streptococci* are found. The eyelids are inflamed upon their borders, the cilia gathered in little tufts by the formation of small pustules at their bases, the conjunctiva is injected and tear-soaked, and there is a somewhat gummy discharge. This form of conjunctivitis may be associated with hypopyon and iridocyclitis, and is a complication which must be removed prior to any operation on the eye; otherwise infection of the operative wound is sure to follow.

The *treatment* requires that the lacrimal passages shall be rendered patulous, in addition to the ordinary remedies suitable for chronic conjunctivitis and ulcerated blepharitis.

Lithiasis conjunctivæ is a troublesome condition caused by a calcareous degeneration of inspissated secretion in the acini of meibomian glands. It is more commonly seen in elderly people than in young subjects, especially if they are gouty. On everting the lids, numerous small, yellowish-white concretions will be seen, distinctly gritty to the touch. These act like so many foreign bodies and produce considerable irritation and pain. They should be distinguished from concretions, more superficially located, which develop within new formed glands in the conjunctiva (Fuchs).

Each concretion should be removed with a fine needle, the conjunctiva having first been rendered insensitive with cocain.

Toxic conjunctivitis includes those forms of conjunctival inflammation caused by certain chemicals and local irritants, by insects, and by the prolonged use of the mydriatics (notably atropin) and the miotics.

Atropin conjunctivitis occurs at all ages, but is commonest in old persons. Sometimes it appears after only a few drops of the solution have been used, but usually not until the drug has been employed for a long time. It has been attributed to impurities in the drug, to the existence of free acid, to the presence of a fungoid growth, and to idiosyncrasy. In a number of instances arthritic history has been obtained (Collins). The disease usually appears in the form of follicular granulations, sometimes associated with much swelling of the lid and eczema of the surrounding tissue. (See also page 235.)

Eserin, hyoscyamin, duboisin, and homatropin less commonly cause this affection, and the same disorder has been reported as the result of the prolonged use of cocain.

Conjunctivitis occurs among those who work in anilin dyes, and from chryosphanic acid, when this has been used as an ointment in skin affections, and may be caused by artificial fertilizers. *Chrysarobin*

conjunctivitis (photophobia, lacrimation, and blepharospasm) may, according to Igersheimer, be associated with gray deposits in the cornea, resembling superficial punctate keratitis.

Conjunctivitis from electric welding, ascribed to ultraviolet rays, from hydrogen sulphid, from the dust from certain kinds of wood, especially satinwood and East India boxwood, and from tar, pitch, mercury, and silver fulminate has often been observed. The conjunctival inflammation thus produced as well as that caused by some of the other substances named may be called *industrial conjunctivitis*. Podophyllin coming in contact with the eye may produce intense congestion, edema of the lids, and infiltration of the cornea (C. Chiari).

Conjunctivitis caused by caustics, acids, and other strong irritants is elsewhere considered. Workers with *x*-rays are subject to a severe and at times intractable form of conjunctivitis (see page 586). Conjunctivitis may follow the stings of flies and other insects, and has been described as due to the presence of larvæ in the conjunctival sac (*larval conjunctivitis*). *Parasitic conjunctivitis* due to one of the groups of the higher fungi has been reported by A. J. Smith, C. M. Hosmer, J. T. Carpenter, Jr., and W. C. Posey. Violent conjunctivitis may be caused by the venom of certain serpents, by eel-blood, and contact with ascarides. Some persons quickly acquire a sharp conjunctivitis if they come near horses, for example, in driving, or if they stroke the fur of a cat or work among certain flowers, notably the primrose, or if plant hairs gain entrance to the conjunctival sac.

Traumatic conjunctivitis includes among its etiologic factors some of the irritating substances described in connection with toxic conjunctivitis. It also arises frequently as the result of wounds of the conjunctival membrane, of the entrance of foreign bodies and of contact with dust-laden atmosphere.

Provoked conjunctivitis is a term applied to various forms of conjunctival inflammation artificially induced by soldiers or recruits desiring to escape military service. The late war furnished many examples of these conditions. The conjunctival lesions were called into existence by the introduction of irritating substances, for example, iodid of mercury, sulphate of copper, tobacco, powdered ipecacuanha, and oil of cloves. Bacteria were rarely present in the secretion which contained quantities of epithelial cells. The lower conjunctival sac was chiefly and sometimes solely affected.

The **treatment** in general demands the removal of the cause, and in atropin conjunctivitis applications of tannin and glycerin and of an alum crystal are useful. In some instances the author has found a 1 per cent. solution of creolin of service. A bland ointment for the irritated cutaneous surface and the ordinary antiseptic lotions are indicated. Boric acid lotion, physiologic salt solution to which may be added holocain (1 per cent.), after the cause has been eliminated, are useful in the treatment of traumatic conjunctivitis.

Poisonous Gas Conjunctivitis.—Exposure to various types of poisonous gases during the late war brought about the following ocular conditions·

Lacrimatory gas caused burning pain, profuse lacrimation, chemosis, swelling of the lid borders, and erythema of lid skin. Occasionally a fine exfoliation of the epithelium of the cornea at its periphery was evident, but these lesions did not extend and mild cases quickly recovered.

Phosgene gas was not conspicuous in producing ocular disorders, but conjunctivitis of various types was observed, occasionally of a mucopurulent type.

The lesions of mustard gas (dichlorethyl sulphid) resembled those of a chemical burn, and were slight, moderate, or severe in their manifestations. Even in the mild cases there were lacrimation, spasmodic closing of the lid, and erythema of their surfaces.

In severe cases the lids were pressed tightly together, were greatly reddened, and often covered by bullæ. The conjunctiva, always intensely injected, became chemotic, especially its upper and lower folds, and not infrequently an area of solid white edema formed in the palpebral fissure, suggesting somewhat the appearance produced by a nitrate of silver burn.

In mild types of the affection, the corneal epithelium was roughened and slightly eroded and it stained with fluorescin; in more severe types grayish areas were noticeable in the roughened cornea (orange skin cornea), and in very severe types the cornea was traversed by a white band in the area of the palpebral fissure. Secondary infection of the cornea was common, especially if the eye had unfortunately been bandaged; conjunctivitis developed, and sometimes severe ulceration, keratomalacia, and even panophthalmitis.

The original conjunctival congestion often was associated with an injection of the ciliary type; slight iritis has been described (Teulières) and even edema of the optic nerve, optic nerve atrophy, and neuroretinitis. It is doubtful, however, if fundus lesions were ever directly due to mustard-gas poisoning.

The mild or benign cases recovered usually within two weeks; in moderately severe cases, which formed the greatest number, the duration of the affection was about six weeks; the white edema disappeared slowly, being replaced by a red area, and often several months elapsed before the soldiers thus affected could return to duty; sometimes the lesions, after subsidence of the white edema, resembled episcleritis (Lister).

Considering the severity of the burn, it is not a little remarkable that comparatively few permanent visual disabilities were noted, and only comparatively rarely total loss of the eye or eyes (keratomalacia, panophthalmitis). In several soldiers observed by and under the care of the author the ultimate results were deeply hazed, moderately vascularized corneas, the epithelium and deeper structures being involved, and vision reduced to hand movements; in one case both corneas were staphylomatous. In some patients, although the corneas were unaffected, the bulbar as well as the palpebral conjunctivas remained for long periods of time injected; exacerbations were common, and there was continuous dread of light.

Treatment.—The most useful collyrium was a 1 per cent. solution of bicarbonate of soda; later boric acid lotion was useful, and liquid albolene was of advantage, but vegetable oils, for example, castor oil, were not satisfactory. In the presence of ciliary congestion and contracted pupil atropin mydriasis was necessary and secondary conjunctivitis was advantageously treated with argyrol. The management of corneal ulcers did not differ from that of civilian practice. Bandaging the eyes was detrimental and was often the cause of secondary infections. Eye shades or dark glasses were required, but it was important not to keep the patients any longer than absolutely necessay in the hospital in order to check the tendency to neurasthenia.

Much work in experimental mustard-gas conjunctivitis has been done, and Warthin recommends a $\frac{1}{2}$ of 1 per cent. solution of dichloramin-T in chlorosone as a lotion. The pathologic examination of gassed eyes shows changes in the corneal epithelium, substantia propria, and in the conjunctiva. The whole subject of the ocular lesions produced by posionous gases has been particularly well studied in this country by Dr. George S. Derby.

Conjunctivitis Nodosa (*Ophthalmia Nodosa; Pseudotuberculosis of the Conjunctiva* [Wangenmann]).—This disease is caused by the irritation of caterpillar hairs which have lodged in the conjunctiva, cornea, or iris, and was first described in 1883 by Pagenstecher. In addition to conjunctival congestion and pericorneal injection, the disease is characterized by a number of grayish or yellowish semitransparent nodules, which are located in the conjunctiva and episclera, the most usual situation being the ocular conjunctiva between the lower border of the cornea and the fornix. In a case studied by the author and E. A. Shumway, 27 such nodules could be differentiated, those directly in the center of the collection being somewhat confluent and assuming a crescentic and circular appearance. The lesions strongly suggest tubercle of the conjunctiva; and, indeed, the disease is called pseudotuberculosis of the conjunctiva by some authors. The center of each nodule usually contains a caterpillar hair, and is surrounded by round-cells, giant-cells, and externally by spindle-cells and a capsule. It is not definitely decided whether the irritation is a mechanical one, or whether it is due to some constituent of the hairs. Not only is the conjunctiva affected, but the hairs may penetrate the cornea, enter the iris, and there form the nodules which have been described. It is probable that they may even reach the choroid. The disease is generally caused by certain species of caterpillar, particularly Lasiocampa, or Bombyx (*B. rubi*, *B. pini*), Liparis (*L. monacha*, *L. dispar*), etc. In the case studied by the author and Dr. Shumway the hairs of the *Spilosoma virginica* were identified. This subject has been elaborately studied by Teuschlaender and by Walter Parker in this country. The *treatment* should consist in excision of the conjunctival nodules and the ordinary remedies for conjunctivitis. A somewhat similar disease, clinically resembling trachoma, has been described by Markus as the result of the implantation of plant hairs.

Xerophthalmos (*atrophy of the conjunctiva; xerosis*) is the name employed by systematic writers to describe a dry, lusterless, and shrunken appearance of the conjunctiva, and is recognized under two forms—*parenchymatous* and *epithelial.*

The former type results from cicatricial changes which involve the deep layers of the conjunctiva; the sulcus is obliterated; the lids, in severe cases, are attached to the eyeball and the cornea is opaque. The surface of the conjunctiva of the lids is smooth, dry, and almost leathery to the touch. Granular and diphtheritic conjunctivitis, pemphigus, and essential shrinking of the conjunctiva are the causes of the disorder.

Treatment is of little avail, but some comfort may ensue by instilling glycerin and water or by the local use of an emulsion of cod-liver oil.

In the *epithelial type* the exposed ocular conjunctiva becomes dry and has a lack-luster appearance; cheesy flakes form, and the membrane is greasy and thrown into folds. A short bacillus (*xerosis bacillus*) has been found in the secretion of these cases, but its pathogenic character is doubtful. This form of xerosis sometimes occurs in epidemics, associated with night-blindness, and is seen among people of poor nutrition—for instance, during prolonged fasts—or among those whose eyes have long been exposed to sunlight. It is also one of the symptoms of keratomalacia in infants. According to Stephenson, the disease is not rare. Night-blindness is not always present, but usually there are signs of torpor of the retina, with contraction of the visual fields and reversal of the red and green fields (see also page 584).

The *treatment* demands a nutritious diet, a soothing collyrium, dark glasses, and removal from the surroundings which have caused the difficulty.

Amyloid disease of the conjunctiva is a rare disorder, mainly observed in Russia and Galicia, in which pale, yellowish, wax-like, friable masses appear, first in the retrotarsal folds; later the palpebral and bulbar conjunctiva and the tarsus are involved. The disease, essentially chronic in nature, may last for years. Although amyloid degeneration of the conjunctiva may follow trachoma, this disease, as Raehlmann pointed out, is not its cause; it may arise in eyes otherwise healthy. Herbert has described hyaline or, as he prefers to call it, *colloid* degeneration of the conjunctiva, and in this country it has been investigated by Bull, Trout, and Bedell.

Extirpation of the lesions is the proper mode of treatment. Their structure is analogous to lymphoid tumors in which a hyaline degeneration may be found, and which, in all probability, is an antecedent condition (Raehlmann, Kubli). But, according to Fuchs, *hyaline degeneration of the conjunctiva* may be distinct from amyloid degeneration. The diagnosis can be made with certainty only by submitting the tissue to the iodin test.

Conjunctivitis Petrificans.—In this rare disease, described by Leber in 1895, a number of irregular white, opaque spots appear in the

conjunctiva, which are slightly elevated above the surface and covered by epithelium. The surrounding conjunctiva is reddened and somewhat inflamed, and any portion of it may be affected, and in advanced stages the disease may spread to the bulbar conjunctiva and the tissue of the lids. The disease may assume a recurring as well as a spreading nature, and in any event is a chronic one, and may last for months or even years. Nothing is known of the etiology of the disease, the white spots consisting of deposits of lime associated with an organic base. All cases thus far reported have occurred in young females, and it is possible that some of the instances recorded represent examples of "provoked conjunctivitis," inasmuch as Sidler-Huguenin observed a case of this character in a hysteric girl who produced the lesions by putting lime into the conjunctival sac. It did not differ from the type ordinarily described.

Pterygium is a peculiar, fleshy growth, consisting of hypertrophy of the conjunctiva and subconjunctival tissue. One or both eyes may be affected. Its most usual situation is at the inner side of the eyeball, corresponding to the course of the internal rectus muscle; more rarely it develops at the outer, and very exceptionally at the upper or lower, part. When the fan-shaped expansion arises from the semilunar fold and caruncle, it converges as it approaches the cornea, the center of which it rarely passes.

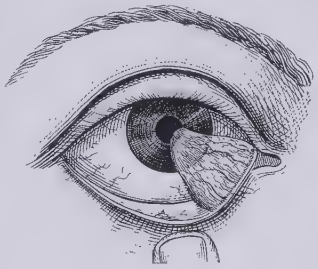


FIG. 117.—Large pseudopterygium, the result of a lime-burn.

The growth is comparatively uncommon in young subjects, the average age, according to Fuchs, being about forty-eight, although it may develop at a much earlier period of life. According to Robert Thompson, of Australia, and John McReynolds, of Texas, in their regions the affection is not infrequently encountered in young subjects. Thompson's youngest patient was only fifteen years old. The theory advanced by Arlt, that ulceration at the margin of the cornea should be regarded as the primary cause of the affection, is no longer tenable. According to Fuchs, pterygium is a development from a pinguecula, and like it, save in exceptional cases, belongs to the so-called senile changes in the eye. As the pterygium develops, the characters of the pinguecula disappear. Exposure to dust, smoke, wind, and heat is the exciting cause, according to McReynolds, who has often seen pterygia both at the inner and the outer canthus. *Pseudopterygia* may result from blennorrhea, burns or erosions of the corneal surface, the thickened conjunctiva becoming attached to the corneal lesion. Occasionally a vascularized tissue spreads gradually over the surface of the cornea, which, clinically, resembles a pterygium except in its shape and disposition.

The **treatment** consists in excision, transplantation, strangulation by means of ligatures, or evulsion (see page 716).

Pinguecula is a small, yellowish elevation situated in the conjunctiva near the margin of the cornea, and usually at the inner side. It has the appearance of fatty tissue, but is a hyaline degeneration of the connective-tissue fibers of the subconjunctival tissue, and should be regarded, according to Fuchs, as the first stage in the development of a pterygium. It may be excised and the conjunctival wound closed with a silk suture.

Abscess of the conjunctiva is a rare condition, in which a localized area of suppuration appears in the subconjunctival tissues. It may develop in children whose nutrition is greatly depressed, and is sometimes the sequel of a wound. *Ulcers of the conjunctiva* are occasionally seen and may be severe enough to destroy the tarsus (Cailloud). Widmark and T. Harrison Butler have described a form of conjunctival disease characterized by congestion of the inferior tarsal conjunctiva and of this membrane in the lower epibulbar expansion. Part or all of the affected area stains with fluorescein. Slight stippling of the cornea may be present. Brow pain and occipital headache may precede the inflammation. Massage with a 1 per cent. ointment of yellow oxid mercury, according to Butler, is the best method of treating *Widmark's conjunctivitis.*

Ecchymosis of the Conjunctiva.—This is an extravasation of blood beneath the conjunctiva, the meshes of the connective tissue being filled with blood-clot, and occurs as the result of an injury, or of an operation, for example, tenotomy, or from some violent, straining effort—*e. g.*, during a paroxysm of whooping-cough or a convulsive seizure, and may be due to fracture of the base of the skull. It may arise without obvious cause, especially in elderly persons, and has been seen in young girls at the time of the menstrual epoch. Its occurrence during severe conjunctival inflammations has been described. *Recurring subconjunctival hemorrhages* are important indications of chronic nephritis and arteriosclerosis. They also occur in diabetes. Ordinarily, subconjunctival hemorrhage subsides by absorption and requires no treatment. *Hemorrhage from the conjunctiva* usually results from an injury or wound, sometimes from the application of an irritant, *e. g.*, nitrate of silver (the author has reported one case of alarming hemorrhage from the conjunctiva following Credé's prophylaxis); but may also appear spontaneously during infectious fevers and in connection with menstruation. It has been noted in newborn children.

Chemosis (*edema*) **of the conjunctiva** occurs where the connective-tissue layer is distended with serum, and is often associated with an inflammatory exudation. It is generally a symptom of some other disease—for example, acute conjunctivitis, choroiditis, iritis, sinusitis, tenonitis, or orbital cellulitis. *Angioneurotic edema* of the conjunctiva, with swelling and hyperemia, may appear without any apparent cause and with marked suddenness. In paralysis of the exterior straight muscles the overlying conjunctiva is often decidedly edematous, and may be an early symptom of this condition. Chemosis of the conjunc-

tiva following the use of iodid of potassium has been reported by the author, and it may succeed a general outbreak of urticaria.

Treatment.—The swollen tissues may be incised and an astringent lotion prescribed.

Emphysema of the conjunctiva consists in a distention of the connective-tissue spaces with air, and occurs under the same conditions which occasion this accident when it involves the eyelids.

Lymphangiectasis of the conjunctiva is a development of small blisters in the conjunctiva, filled with semitransparent fluid, and usually gathered together in masses. These are situated superficially, and readily move with the conjunctiva over the subjacent tissue. (An interference with the natural lymph flow and consequent distention of the lymph-spaces is the probable explanation of their appearance. The affection is said to be most frequent in children, but may occur at any age.) Spontaneous disappearance is the common outcome, but, if need be, the small blisters may be incised.

Syphilis of the Conjunctiva.—Chancres may develop on the upper or lower culdesac, and even upon the ocular conjunctiva, as primary affections, and not only as extensions from the lids. A few instances of soft chancre have been described.

As manifestations of general syphilis, ulcerated papular syphilids and gumma of the conjunctiva have been recorded. Mucous patches occasionally develop on the conjunctiva. Finally, there is a type of inflammation called *syphilitic conjunctivitis*, which appears as a stubborn catarrh, or in the form of granulations similar to trachoma follicles, in an anemic and rather colloid-looking conjunctiva. The disease is not amenable to local treatment, but disappears under antisyphilitic remedies. Conjunctival lesions in hereditary syphilis are uncommon; papular syphilids and gummas have been described.

Tumors and Cysts of the Conjunctiva.—As congenital forms, translucent cysts, angiomas, cavernous angiomas, lymphangiomas, dermoid growths (see page 317), and pigment spots have been described. *Moles of the conjunctiva* are usually deeply pigmented (occasionally they have a gray or pinkish color); rarely they spread over the tissue without involving the deeper layer. Pigment patches, especially near the limbus, are not uncommon in persons of dark complexion and have no pathologic significance. Angiomas may be situated on the palpebral conjunctiva, the bulbar conjunctiva, the fornix, or the plica. Usually congenital in origin, they may arise in later life. A nevus may be the starting-point of a sarcoma. Pigment spots, after healing of variolous pustules, have been described. *Nævus pigmentosus* also occurs (Wintersteiner) and may give rise to sarcoma. Pigment patches on the ocular and palpebral conjunctiva have been observed—*melanosis of the conjunctiva.* In a patient under the care of the author there was marked bilateral pigmentation in both conjunctival sacs, with patches of pigment in the semilunar folds and caruncles. He was one of a family with this peculiarity, which had existed in a number of generations.

Cysts of the conjunctiva may appear in the bulbar conjunctiva, in the

palpebral conjunctiva, and in the fornix. Parsons describes the following varieties: Retention, lymphatic, traumatic, parasitic, and congenital cysts. This materially simplifies Cirincione's elaborate classification. Retention cysts, often seen in the region of the retrotarsal folds, are small, oval, clear bodies. They develop in new-formed glands as the result of inflammation, in the so-called Henle's glands, and more rarely in Krause's glands. They may also rise from the accessory lacrimal gland. Lymphatic cysts appear in the bulbar conjunctiva, and represent dilatations of lymphatic vessels (lymphangiectasis and lymphangioma; see page 257). Usually multiple, they occasionally appear as isolated, yellowish cysts. *Traumatic cysts* arise as the result of a conjunctival wound or injury, and sometimes are *implantation cysts*—that is, through the wound epithelium from the skin, cilia, etc., gains entrance, degenerates, and produces the cyst. *Parasitic cysts* are usually due to

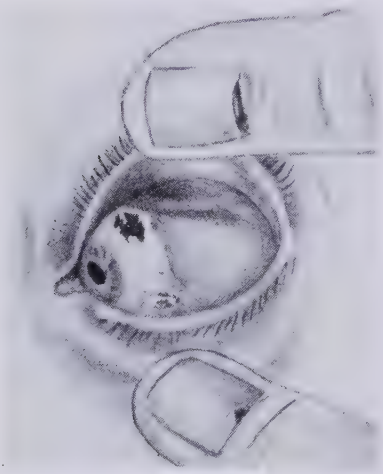


Fig. 118.—Epibulbar lipoma; pigment areas on sclera (from a patient in the University Hospital).

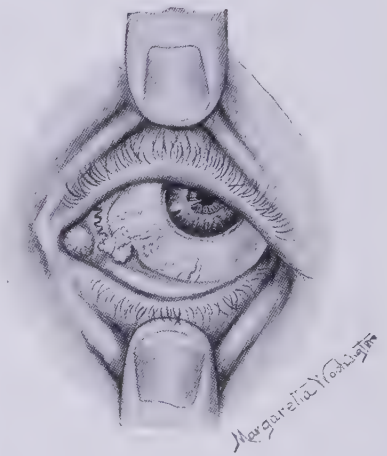


Fig. 119.—Papilloma of the conjunctiva (from a patient in the Philadelphia General Hospital).

the presence of a cysticercus, and appear as large, yellowish vesicles. A white spot in the wall may indicate the situation of the embryo. Filiariæ may also cause conjunctival cysts. To a growth situated near the corneal margin, semitranslucent in color, and often of congenital origin, Parinaud gave the name *dermo-epithelioma*. For this title Oatman preferred the name "epithelial cystoma of the conjunctiva," as he believed it represented a transitional stage in the development of the epithelial cyst.

Among the benign tumors, dermoids (see page 317), lipoma, fibroma, osteoma, granuloma, adenoma, hemangioma, and papilloma have their habitat upon the conjunctiva. Lipomas and lipomatous dermoids are found between the superior and the external rectus. Double symmetric lipodermoids at the inner commissure, of congenital origin, and arising

from the plica or caruncle, have been recorded (Müller, Vossius). Fibromas are either hard or soft, and appear in the form of polypi. Soft fibromas occur chiefly in the fornix or palpebral conjunctiva, and are

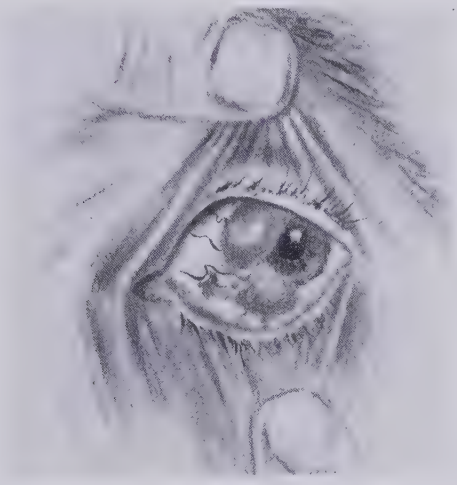


FIG. 120.—Papilloma of the corneoscleral border, followed nine years after its removal by an epibulbar carcinoma.

often highly vascular. Hard fibromas are less common, and arise from the palpebral conjunctiva or caruncle. Bone formation usually occurs between the margin of the cornea and the commissure. Papillomas are either pediculated or sessile, and histologically resemble the structure of the papillæ. They may arise from the conjunctiva or plica, and are often multiple. Ordinarily benign, they may undergo carcinomatous degeneration and infiltrate the eyelids (Risley and Shumway) or give rise to an epibulbar carcinoma (Figs. 120, 121). Papillomas have been confounded with masses of granulation tissue arising from wounds—*e. g.*, after strabismus operations, and with angiosarcomas. Adenomas may originate on the conjunctival surface of the lids from Krause's and Moll's glands, and from the meibomian glands. They may develop into malignant growths —*adenocarcinomas.*

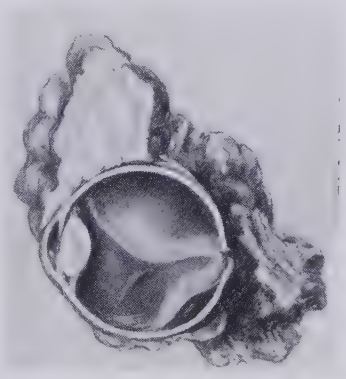


FIG. 121.—Epibulbar carcinoma and adjacent orbital tissue, nine years after removal of a papilloma (see Fig. 120).

Treatment.—Usually the growths described can be readily excised, and the edges of the wound may be united with fine sutures. In simple cysts, cutting away the anterior wall is generally sufficient to cause

a cure. Nevi have been treated with applications of ethylate of sodium (Snell). The removal of papillomas should be thorough, as they have a tendency to undergo carcinomatous degeneration.

The malignant growths include epithelioma and sarcoma.

Epithelioma may occur as a primary growth upon the ocular conjunctiva, especially at the limbus corneæ, and rarely appears before the fortieth year of life. Commonly situated at the outer side, it may also appear on the nasal side, and in rare instances has surrounded the entire cornea or encircled the globe (*peribulbar epithelioma*).

An epitheliomatous or carcinomatous growth usually first manifests itself as a small, reddish elevation surrounded by injection. Generally the growth is slow; its base is broad and attached to the underlying tissue; rarely a large fungous mass is formed. The tumor is com-

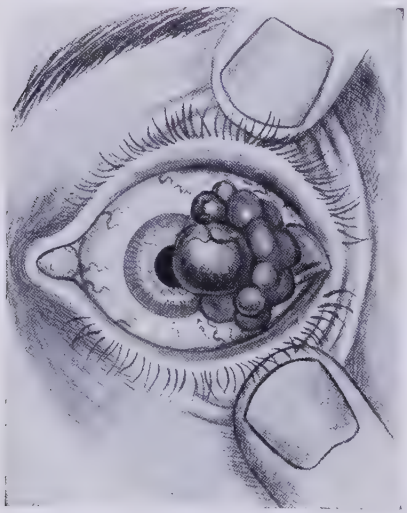


FIG. 122.—Sarcoma of corneoscleral junction (from a patient in the Jefferson College Hospital).

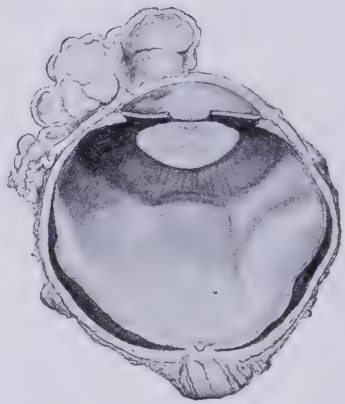


FIG. 123.—Section of eyeball (see Fig. 122) with sarcoma of corneoscleral junction.

posed of proliferating masses of epithelium which proceed from the surface epithelium, and which are separated into alveoli by a connective-tissue stroma. As a rule, the substantia propria of the cornea is infiltrated, and if the growth involves the eyeball, it does so along the perivascular and perineural lymph-sheaths. Conjunctival epithelioma may be pigmented (*melanocarcinoma*).

Sarcoma of the conjunctiva arises at the limbus (*epibulbar sarcoma*), its subjects generally being past middle life, in the form of a reddish-white or brownish-black growth, usually overlapping the cornea, but not often involving its structure. Both pigmented and unpigmented varieties occur, the former being the more frequent. The tumors may grow rapidly and reach a large size. They are composed of round- and spindle-cells, and may have a markedly alveolar arrangement (*alveolar*

sarcoma); sometimes the cells are distinctly epithelioid in type (*endothelioma*). Epibulbar sarcomas may develop from collections of pigmented cells on the conjunctiva (*pigment spots, melanomas*). Rarely they invade the interior of the eye. Sometimes they are multiple. Sarcomas of the palpebral conjunctiva and fornix and diffuse melanotic sarcomas have been recorded. *Angiosarcomas of the conjunctiva* arise from a proliferation of the adventitia of the blood-vessels, and in their growth, like fibromas, they thrust the epithelial covering in front of them. They have been mistaken for papillomas.

Prognosis and Treatment.—Epitheliomas vary in malignancy; but they tend to recur even when superficial. Occasionally it may be proper to remove the growth, with the expectation of saving the eyeball; but if it is involved, complete removal of the globe is indicated. Should the growth be excised the operation should be followed by radium applications or these may be used without a primary excision. Collins reports the dissipation of a growth of this character after a single application of radium bromid (10 mg.) (see also page 191). Epibulbar sarcomas have been removed and the eyeball preserved; but Verhoeff and Loring regard them as highly malignant, inasmuch as there is a history of recurrence in fully 80 per cent. of the cases, and recommend that the eyeball should be removed at once. Excision of these growths is practicable in the early stages without sacrificing the eyeball. This excision followed by radium treatment may achieve satisfactory results. To the method of treating epibulbar growths by means of "electric desiccation" reference has been made elsewhere (page 191).

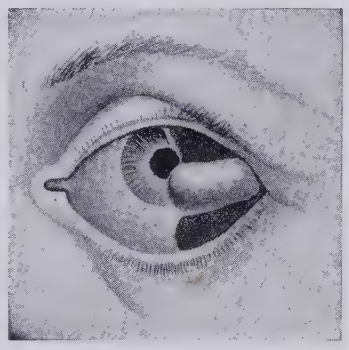


FIG. 124.—Sarcoma of the conjunctiva (from a patient in the Philadelphia General Hospital).

Lepra.—According to Lopez, the chief alterations in the conjunctiva produced by leprosy are anesthesia, inflammation, pterygia, and tubercles. The anesthesia of the conjunctiva probably determines the chronic conjunctivitis, which is common. Pterygia are frequently observed, and are caused by the action of external irritants upon the ocular conjunctiva, which has become insensitive under the influence of the disease.

It is convenient in this place to refer to the effect of *leprosy upon the cornea*, in which the lesions are frequent and varied. The tubercles which form in the conjunctiva are apt to attack the corneoscleral margin, but may involve the cornea exclusively. A late manifestation of the disease is an inflammation of the cornea known as *leprous keratitis*, which somewhat resembles interstitial keratitis.

Lupus occurs as a primary disease, or extends to the conjunctiva from the surrounding integument. It appears in the form of red, granu-

lar patches placed upon an ulcerated base. As the same microbe is the cause of lupus and tuberculosis, any difference existing in the two diseases occurring in this situation must rest upon the clinical appearances, the lupus spot showing healing in one direction and active ulceration in another. Those cases in which the disease has spread from the lid to the conjunctiva have especially been classified as lupus.

Tubercle of the conjunctiva occurs as a *primary* and as a *secondary* affection.

Primary tuberculosis of the conjunctiva is rare, but a certain number of instances are upon record in which there was an absence of evidence of tuberculosis elsewhere, and in which there was no reappearance of the disease locally, or in distant organs, after its removal. Villard maintains that in 60 per cent. of the cases he has analyzed no initial tuberculous lesion was discovered, and therefore he does not believe in its endogenous origin.

As a secondary affection it has usually appeared in association with nasal and laryngeal tuberculosis.

According to Eyre, who adopts Sattler's classification, the disease may appear in one or other of the following manifestations: (1) One or more miliary ulcers which usually caseate; (2) grayish or yellowish subconjunctival nodules which resemble the sago granules of trachoma; (3) florid hypertrophied papillæ and rounded, flattened outgrowths of granulation tissue; (4) numerous pedunculated cock's-comb excrescences; (5) a distinctly pediculated tumor. The ulcers have uneven and slightly raised edges, and their floors have a yellow or sometimes a lardaceous appearance.

There are thickening of the lids, dark red swelling of the conjunctiva, considerable discharge, and occasionally tumefaction of the tear-sac. The preauricular and submaxillary lymphatic glands of the same side are enlarged. Pain is not considerable unless the ulceration involves the bulbar conjunctiva and cornea or extends to the lids.

The disease should be distinguished from trachoma, epithelioma, and syphilitic ulceration.

Diagnosis.—In any suspected case the real nature of the affection may be decided by excising a portion of the diseased tissue, submitting it to microscopic and bacteriologic examination, or by submitting the patient to a test with tuberculin, or by implanting a portion of it in the anterior chamber of a rabbit's eye. It is not always possible to demonstrate the presence of tubercle bacilli.

In trachoma the lymph-glands are not involved and the discovery of the Prowazek bodies (see Fig. 111) would tend to establish the diagnosis.

Epithelioma is excluded by the age of the subjects, tuberculosis almost invariably occurring in young persons; that is, those under the thirtieth year of life.

Prognosis.—This depends upon whether the disease is primary or secondary. In order to prevent general infection it is important to eradicate the local lesion. Sight may be destroyed by involvement of the cornea.

Treatment.—The diseased tissue may be removed with a knife or curet; the galvanocautery has been recommended. The subsequent treatment should include the use of a collyrium of bichlorid of mercury, and iodoform or aristol powder. Injections of *tuberculin TR* are advisable, and, according to Ormond and Eyre, represent a treatment far superior to excision and scraping. Stephenson suggests the trial of the *x*-rays. Axenfeld uses a 50 per cent. solution of lactic acid in the treatment of conjunctival tuberculosis; after its application the eye is irrigated with a saline solution.

Pemphigus of the conjunctiva is a rare affection and it is rather uncommon to detect the bullæ in the conjunctiva which are characteristic manifestations of the cutaneous lesions of this disease. Instead of vesicles, membranous exudations, grayish white in color, form on areas of conjunctiva deprived of their epithelium. A process of cicatrization and contraction ensues, fresh gray-coated areas develop in other portions of the conjunctiva to be followed by additional destruction and cicatrization of the involved membrane. According to Michel, the disease may be confined to the conjunctiva, or it may attack not only the conjunctiva but also the mucous membrane of the nose, mouth and pharynx, and the skin. Coincidence of pemphigus of the skin and of the conjunctiva is, however, uncommon.

The course of the disease with its almost invariable recurrences is slow and may extend over months and years (occasionally it is stationary), and is destructive not only to the nutrition of the conjunctiva but later to that of the cornea. The former undergoes the cicatricial change which has been described and the latter becomes opaque and staphylomatous; often the lids and eyeball become adherent (total symblepharon); rarely the cornea may escape for long periods of time. The extreme fetor in pemphigus to which Stieren refers was most evident in a case recently in the care of the author.

Under the name *essential shrinking of the conjunctiva* a condition of atrophy, contraction, and gradual disappearance of the conjunctival culdesac has been described, during which the free borders of the lids become fixed to the ball and the cornea becomes dry and opaque. This probably is a form of pemphigus, but has also been recorded as an essentially distinct process. According to Pergens, essential shrinking of the conjunctiva may be produced by trachoma, psoriasis, xeroderma pigmentosum, ichthyosis, and lupus.

Treatment.—This is practically unavailing. Applications of glycerin and water and other emollients have been employed with the hope of keeping conjunctiva moist, and *x*-rays (Neeper) and thiosinamin (Melville Black) have been advised. Rabbits' conjunctiva and human conjunctiva have been transplanted, but without beneficial results. The author attempted this procedure on two patients without success. The internal administration of arsenic has been recommended and, as some of the patients are also syphilitic, mercury, iodid of potassium and arsphenamin should be tried. E. A. Shumway practised excision of the tarsal cartilages from the greatly thickened upper lids in order

to relieve the pressure upon the eyeball. His operation was followed by marked relief and distinct improvement in the corneal condition. A lotion of resorcin and boric acid (1 gram of each to 920 c.c. of distilled water), recommended by Schamberg, is useful in mitigating local irritation.

Injuries of the Conjunctiva.—(*a*) **Foreign Bodies.**—A small particle of coal, ash, or dust is easily removed if lodged upon the lower portion of the conjunctiva; but if it finds its way beneath the upper lid, and is situated far back under the retrotarsal fold, it may not come into view when the lid is everted unless the fold is pushed into prominence. If the foreign body is attached to the tissues it may be necessary to dislodge it with the point of a needle or with a spud. Cocain, butyn, or holocain render this operation painless.

(*b*) **Wounds.**—These may be part of a serious injury involving the lid or deeper structures of the eye; more rarely they occur as simple lacerations, confined usually to the bulbar portion. In suitable cases, after proper cleansing, the lips of the wound should be drawn together with a few sutures.

(*c*) **Burns.**—These are commonly due to lime (mortar or quicklime), ammonia, molten metals, powder and acids, and are especially serious because of the deformity which the subsequent contraction is likely to produce, or on account of the development of a symblepharon (see page 196). Ulceration of the cornea, hypopyon, and even panophthalmitis may result. The prognosis of such injuries is always grave.

All foreign substances must be removed at once, and if lime has been the injuring agent, this is best accomplished by forcible irrigation of the conjunctival sac with clean water. Schmidt-Rimpler, however, preferred, in these circumstances, thorough cleansing of the eye with oil introduced with a syringe into the culdesac. For acid burns an alkaline lotion is usually recommended. The subsequent treatment consists in the instillation of olive or castor oil, and atropin drops to prevent secondary iritis if the cornea is much inflamed; atropin may be incorporated with liquid vaselin and placed in the culdesac. To prevent the formation of symblepharon the adhesions forming between the bulbar expanse and inner surface should be daily parted by means of a blunt probe. As soon as the wounded surface is clean and granulations are visible an epidermic or mucous membrane graft may be adjusted (Wilder). Denig recommends immediate interference and after removal of the detritus and burned conjunctiva plants a mucous membrane graft. Powder grains are frequently embedded in the conjunctiva; they cannot be picked from their beds; usually the larger ones can be excised.

Affections of the Caruncle.—The caruncle and semilunar fold may be swollen in conjunction with a general inflammation of the conjunctiva, but also may undergo localized enlargement and inflammation, to which the name *encanthis* has been applied, and which is subdivided by systematic writers into an *acute* or *inflammatory* and a

chronic variety. The process may go on to the formation of a minute abscess.

Swollen caruncles are commonly found in patients with eye-strain, especially with imperfect amplitude of convergence. The small body is red, elevated, and angry looking, and injected vessels run from it toward the cornea in the interpalpebral space. The condition might be designated *symptomatic* or *functional encanthis.*

In like manner, temporary irritation of the structure is caused by the lodgment upon it of a foreign body, or by the presence of misplaced cilia which rub against it. The caruncle should be carefully examined when patients complain of irritation, lacrimation, and inability to use their eyes with comfort.

The excessive development of the hairs normally placed upon the caruncle is called *trichosis carunculæ.*

A number of tumors situated upon and growing from the caruncle have been recorded; in general terms they may be of epithelial-conjunctival origin, or constitute mixed types (John Green, Jr.); in two instances the growth proved to be an adenoma (Prudden and Schirmer). Primary sarcoma (Veasey, Snell) and carcinoma of the caruncle (*malignant encanthis*) have been described. Papilloma, dermoids, nevus, fibroma, lymphangioma, epithelioma, cylindroma, angiosarcoma, and lymphosarcoma have also been reported (V. Berl). Cysts, both dermoid and sebaceous, pustules, and concretions of calcium salts have been observed.

Treatment.—Local irritations of this body may be relieved by the direct application of a mild astringent like alum, or soothed by touching it with tincture of opium. Foreign bodies, stiff hairs, and misplaced cilia must be extracted. A tumor should be removed by excision.

Argyria Conjunctivæ (*Argyrosis*).—Long-continued application of solutions of nitrate of silver to the conjunctiva may be followed by a brownish discoloration of this membrane. For this reason it is inadvisable to allow patients to use at home even a weak collyrium of this drug. The same discoloration follows the injudicious use of protargol, argyrol, and largin; indeed, these drugs produce the stain more quickly than nitrate of silver, even, it is said, after a few weeks of their employment. The coloration is due to staining of the elastic fibers; the epithelium is free from pigment. Argyrosis from nitrate of silver is practically irremediable, although the use of dionin is said to decrease its intensity (Lebensohn). Argyrosis from argyrol decreases after a discontinuance of the drug (Krauss). A collyrium of iodid of potassium (0.5 per cent.) may be tried.

A yellowish-brown discoloration of the conjunctiva, known as *siderosis conjunctivæ,* due to the prolonged use of sulphate of iron, has been reported.

CHAPTER VII

DISEASES OF THE CORNEA

Under the general term *keratitis* are included divers forms of inflammatory affections of the cornea, to which, according to the type, certain well-marked stages belong: cellular *infiltration* in the layers of the cornea going on either to absorption or to the formation of pus; loss of the substance of the cornea lying over the infiltrated area, and the development of an *ulcer;* loss of the transparency of the superficial corneal layers over an infiltrated area, which has been converted into pus and created an *abscess,* with the final destruction of these layers by the development of the abscess; the appearance of *vessels in the cornea;* and the process of *repair* after loss of substance, or the period of *cicatrization.*

Keratitis may be classified as *primary* if it develops in an eye previously normal, due to a cause which acts directly upon the cornea, and as *secondary* if it is dependent on disease of some other part of the eye, for instance, the conjunctiva. Should the infection come from without, the keratitis is *ectogenous;* if from within, that is, by way of the blood streams, it is *endogenous.* A convenient classification from the clinical standpoint adopted by systematic writers is *ulcerative* (or *suppurative*) and *non-ulcerative* (or *non-suppurative*) keratitis.

In many types of keratitis both suppurative and non-suppurative, the corneal lesion is not alone in evidence; iritis, iridocyclitis, exudation into the anterior chamber or hypopyon are frequent complications. The usual subjective symptoms include diminution of vision, pain, photophobia, excessive lacrimation, and blepharospasm.

Although it is customary to divide the many types of corneal inflammation into suitable groups, it is by no means possible to refer the disease in each instance to one or other of these divisions.

Phlyctenular Keratitis or Keratoconjunctivitis (*Eczema of the Cornea*).—This disease is characterized by the formation of single or multiple phlyctenules on some portion of the cornea, and is accompanied by dread of light, excessive lacrimation, and blepharospasm.

Causes.—The disease is commonly seen in so-called scrofulous subjects, rarely before the first year of life, most frequently in children before the age of puberty, and less commonly in adults. It often is secondary to phlyctenular conjunctivitis or is associated with it (see page 231). Enlarged lymphatic glands, prominent and swollen lips, and diseases of the joints and bones may be present.

This form of keratitis is in close connection with obstructive (adenoid vegetations) and inflammatory diseases of the nasal passages, and an infectious rhinitis is frequently an associated disorder; ethmoiditis

and infected tonsils may be present. The clinical connection between this disease and eczema is intimate, and eczema of the face, scalp, and around the nares is often an accompanying condition. The affection not uncommonly follows in the wake of measles or other acute exanthemata, and is distinctly under the influence of climate, being aggravated in warm and moist weather. Tuberculosis of the lymphatic glands is present in fully one-half of its subjects, and the evidence is daily increasing that phlyctenular keratitis is closely connected with tuberculosis and probably caused by it. A large number of the subjects of phlyctenular disease (88 per cent., G. S. Derby, Stock; 90 per cent., Gibson) react to tuberculin—a suggestive fact, although it does not prove that tuberculosis is the cause of the disease. Phlyctenules not infrequently have developed as the result of the Calmette reaction and have also followed subcutaneous injections of tuberculin. W. Stanley Gibson, who has produced phlyctenules experimentally in tuberculous rabbits, as the result of a careful examination of 92 patients with phlyctenular keratoconjunctivitis maintains positively that all the evidence points to tuberculosis as the cause of this disease.

Staphylococcus pyogenes aureus and *albus* are present in the epithelium of the affected regions; but these organisms are not found in fresh phlyctenules. *Tubercle bacilli* have not been discovered. The exact cause of the ocular lesions, or phylctenular eruption, has not been determined.

Symptoms.—The phlyctenules, which consist in the early stage of minute subepithelial collections of round cells, appear upon the cornea usually at or near the corneoscleral junction. They vary in size from a poppy-seed to a millet-seed; their tops, at first gray, speedily grow yellow, break down, and form superficial ulcers. They are accompanied by decided local congestion, increased lacrimation, and photophobia.

The palpebral conjunctiva, always hyperemic, may remain translucent and bathed in tears, or the disorder may be complicated with mucopurulent conjunctivitis or phlyctenules scattered over the conjunctiva may be present (page 231).

When the photophobia is severe, the child buries its head deeply in the bedclothes; the lids are spasmodically closed, rendering inspection of the eye difficult, at times well-nigh impossible. The dread of light and the blepharospasm are probably due to direct irritation of the corneal nerves, as Iwanoff found the cellular infiltration situated along their course.

The pustule, when it breaks down, forms a *phlyctenular ulcer.*

This may remain at its original seat near the margin, or creep toward the center of the cornea (*migratory pustule*), followed by a bundle of thickly crowded blood-vessels, and form a special type of corneal inflammation, known as *fascicular keratitis.* The blood-vessels, when the ulcer heals, disappear, but a stripe of opacity remains.

Under the name *phlyctenular marginal keratitis* a variety of this disorder exists, characterized by the development of numerous phlyc-

tenules along the rim of the cornea, giving rise to a process which may cease here, or which, by further invasions, may produce vascular ulcers.

More dangerous than any of the other varieties is the formation of a *single pustule*, just at the corneal border, which speedily ulcerates and is surrounded by a yellow area of infiltration, with a strong tendency to perforate the corneal layers.

If these inflammations recur constantly, the cornea becomes clouded, uneven from loss of epithelium, and covered with numerous superficial vessels, the whole forming the so-called *phlyctenular pannus*.

Sometimes in the middle and deep layers of the cornea extensive gray or yellow opacities may form, which may suppurate, with large loss of tissue, or go on to resorption. These are the so-called *deep scrofulous infiltrations*.

Pathology.—The efflorescence or phlyctenule consists of a collection of lymphoid cells, lying between Bowman's membrane and the epithelium, by the softening of which, as before described, the superficial cells are discharged, and an open, ulcerating surface is exposed. By further degeneration the entire nodule disappears, and the loss of substance is rapidly replaced by epithelium.

Diagnosis.—This presents no difficulties, direct inspection rendering the nature of the disease evident.

Prognosis.—The course varies greatly; in mild cases healing takes place with only a slight loss of substance, and the resulting scar is scarcely discernible.

Not so with the severe forms, in which there has been decided loss of substance and a distinct scar-tissue remains, or in which deep ulceration with perforation occurs, or where constantly recurring ulceration leaves an uneven and roughened surface. In children of the tuberculous type, especially if their surroundings are unfavorable, phlyctenular keratitis is exceedingly intractable.

Treatment.—In order to make a thorough application of the local remedies the child's head should be taken between the surgeon's knees and the lids separated, while the attendant holds the hands and body; the cornea will usually roll out of sight, but gradually may be coaxed into view. Sometimes a lid-elevator is useful, and a few whiffs of ether or chloroform may be necessary.

If much secretion is present, boric acid or physiologic salt solution should be employed, and atropin drops should be instilled with sufficient frequency to maintain mydriasis. Cocain, judiciously used, will allay the photophobia, but its continuous application where corneal ulcers exist is to be deprecated. Holocain is sometimes useful; dionin occasionally seems to act unfavorably in phlyctenular keratitis. An ointment of the yellow oxid of mercury, gr. j to 3j (0.065–3.885 gm.), either with or without the addition of atropin, may be employed, or calomel may be dusted into the conjunctival sac, provided no form of iodin is being exhibited (see page 231). The eyes should be protected if possible by colored glasses and the child encouraged not to bury its head in the bedclothes.

Douching the eyes with cold water will subdue the dread of light, and touching the ulcerated external commissure, which almost invariably exists in these cases, with a crystal of bluestone, as Koller has suggested, helps to relieve the blepharospasm. In severe cases the ulcerated fissure may be incised or the lids may be forcibly separated. No doubt this acts by stretching or rupturing a few fibers at the commissural angle, and relieves the spasm in the same manner as a similar manipulation is efficacious in fissure of the anus.

The best possible hygienic surroundings must be obtained; fresh air and wholesome food are essential. Tea, coffee, and sweets, that is, cake, candy, pastry, etc., must be strictly forbidden. A proper diet in this affection is of paramount importance. Cod-liver oil, iron, especially syrup of the iodid of iron, syrup of hydriodic acid, quinin, often suitably given with pepsin, and arsenic are the most acceptable internal remedies.

The urine should be examined in all these cases; and scrupulous attention paid to the condition of the alimentary canal is an important factor in the treatment. The administration of calomel in small doses, persistently used, is of real value.

If rhinitis is present, a powder composed of equal parts of pulverized camphor, boric acid, and subnitrate of bismuth is useful (Augagneur), especially if the parts are thoroughly cleansed with Dobell's solution before its insufflation into the nasal chambers; powdered iodoform may be used in like manner, but its odor is objectionable, hence nosophen is preferable, and borobismuth ointment is recommended. The affected mucous membrane may be painted with compound tincture of benzoin or sprayed with permanganate of potassium (1 : 5000). If possible, however, this part of the treatment should be confided to an expert rhinologist. Removal of adenoids or of infected tonsils is often urgently needed. That ethmoiditis may be present is well known. The teeth, too often neglected in children with this disease, require special consideration. A patulous condition of the lacrimal passages should be secured.

In stubborn forms of recurring vascular ulcer and deep ulceration, especially in the fascicular type, the use of the actual cautery in the manner later described is productive of excellent results, or the ulcer may be touched with trichloracetic acid. In general terms, the treatment of severe types of phlyctenular ulcer is the same as that recorded on pages 278–280. In phlyctenular pannus *peritomy* is sometimes a useful procedure, and *canthoplasty* may be necessary.

The best results in the treatment of phlyctenular disease are secured if its subjects are treated like other cases of tuberculosis—viz., after the patient is instructed as to general living, proper food, etc., he is visited in his home by one of the class-workers in the social service of the hospital, and is shown how to live and helped to carry out all directions. This method, advocated by George Derby, the author has followed with satisfaction in his hospital patients. Tuberculin therapy is highly commended by some physicians in the therapeutic manage-

ment of this disease; certain observations indicate that it tends to prevent the relapses which are so common in this disorder (page 268). After healing, provided the condition of the cornea permits it, refractive error should be corrected.

In general terms, phlyctenular inflammation of the cornea, which has just been described, is a circumscribed, usually superficial keratitis, and is known under a variety of synonyms—lymphatic, scrofulous, vesicular, fascicular, and pustular—and where it appears in adults it may assume the form of a simple corneal infiltration. It furnishes the greatest number of ulcers of the cornea which are found in early life.

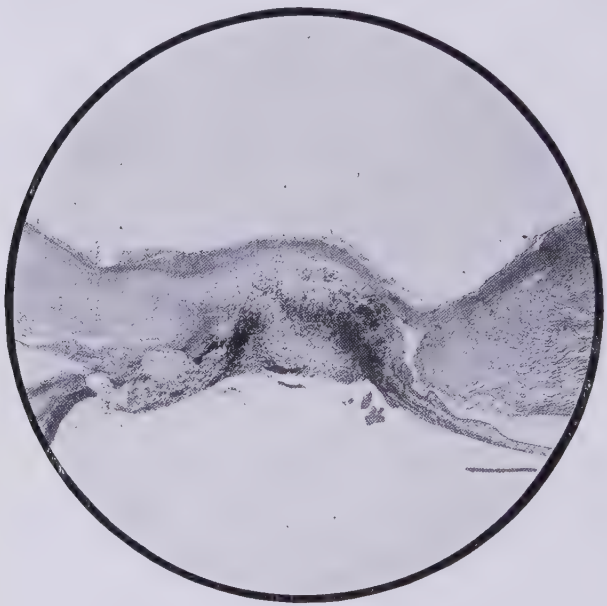


FIG. 125.—Perforating ulcer of the cornea with incarceration of iris (from a photomicrograph).

Ulcers of the Cornea.—If the stage of infiltration fails to terminate in absorption and there is destruction of the overlying corneal tissue, an ulcer results. Surrounding the area of necrotic tissue is a clear space, and beyond this a ring of infiltrating leukocytes which come from the vessels at the edge of the cornea. In favorable cases this necrotic tissue is thrown off, the surrounding cornea clears, the ulcer is covered by a proliferation of the epithelium, and the loss of substance is replaced by connective tissue derived from the fixed cells of the cornea. Where the process is progressive, successive layers of the cornea become involved, the iris and ciliary body are involved, and hypopyon forms (see page 273). If the ulceration is not checked, the cornea is perforated, and inclusion of the iris may result in *staphyloma* (see page 287). If the iris does not prolapse, the perforation may be

closed with a tissue produced by proliferation of the posterior lining endothelium. Bowman's and Descemet's membranes are never reproduced.

In addition to those which have been described with phlyctenular keratitis, corneal ulcers may be gathered into several groups:

1. **Simple ulcer** appears in the form of a small, superficial, gray lesion, associated with slight pericorneal vascularity, and results from the rupture of a phlyctenule ("pimple ulcer") or from trauma.

An ulcer, which, from its situation, is called a *small central ulcer*, may develop as a gray or grayish-white opacity in the center of the cornea, and is not accompanied by much vascularity or dread of light. The elevation is slightly cone shaped until the whitish top breaks down into a shallow depression.

Usually single, this form of ulcer may be multiple, and tends to recur. It is seen in young children who have been poorly nourished and are of a so-called strumous habit. While healing generally occurs with promptness, a permanent opacity may remain, which, from its central situation, may seriously impair vision. The situation of such an ulcer is not always central; it may develop in the cornea at a point midway between the center and the periphery. If neglected, and in patients of poor nutrition, ulcers of these characters occasionally form abscesses of the cornea, or change their type and develop into the following variety:

2. **Purulent** or **deep ulcer** consists of an area of yellowish (purulent) infiltration, surrounded by a zone of hazy cornea, round or irregular in shape, centrally excavated, and with a tendency to travel inward until perforation occurs, but not to extend in a lateral direction. Like all severe types of corneal ulceration, it may be associated with inflammation of the iris and the formation of pus in the anterior chamber; if perforation takes place, an adherent scar or leukoma results.

This ulcer is either *primary*, and sometimes contains a foreign body as its nucleus, or is *secondary* to a violent grade of conjunctival inflammation. The subjective symptoms are pain, browache, congestion, and sometimes, though not necessarily, photophobia.

3. **Indolent ulcer** (*absorption ulcer*) occurs under several forms: (*a*) *Shallow central ulcer*, with slightly turbid base, unattended with any considerable pain or photophobia, essentially chronic in its course, and healing finally with a faintly opaque facet (*faceted ulcer*).

(*b*) *Excavated* or *gouged-out ulcer*, often seen in children, most troublesome because it is rebellious to treatment, has its seat near the corneal margin. It may be entirely overlooked on account of the absence of congestion, and because in appearance it is a small, punched-out excavation with transparent bottom, and free from any opaque surrounding. The floor of the ulcer loses its translucency when healing is about to take place, and a few vessels of repair pass to its margin. To one type of small marginal ulcer, usually unassociated with severe reaction, the name *catarrhal ulcer* is given; it may be accompanied by conjunctivitis.

(*c*) *Reparative ulcers* are seen when, as occasionally occurs in the course of the healing of an ordinary corneal ulcer, this loses its turbidity and assumes a clear, facet-like appearance. These are similar to the absorption ulcers which occur primarily, and which, associated with injection and with local symptoms, may, none the less, extend inward and perforate the cornea.

Indolent ulcers, in general terms, may depend upon some failure in the nutrition of the cornea due to neuropathic disturbance. They are found in anemic and scrofulous subjects, and are seen in cases of chronic trachoma. Central ulcers may also arise in association with Koch-Weeks' and pneumococcus infections. Because of the central situation of the small cicatrix (macula) which remains vision is often greatly disturbed by the production of irregular astigmatism.

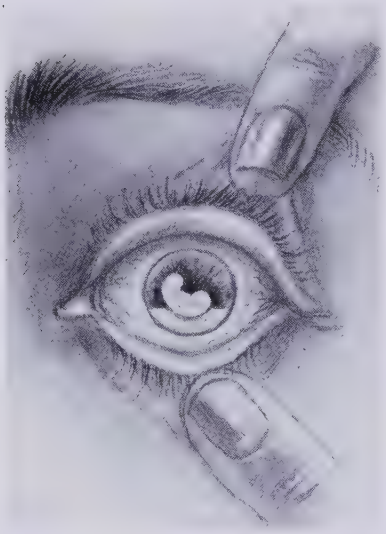


Fig. 126.—Infected ulcer of the cornea, with hypopyon—hypopyon-keratitis (modified from Haab).

4. **Infected or Sloughing Ulcer** (*Purulent Keratitis*).—Ulcers without vessels of repair, which spread widely from one border and readily become complicated with hypopyon and iritis, and which are often the result of a trifling injury, usually affect elderly persons and those whose nutrition is depressed.

The most important type of these is the *acute serpiginous* or *creeping ulcer* of Saemisch; also called the *pneumococcus ulcer* and *ulcus serpens*. In the beginning a nearly central gray area forms, which ulcerates; its margins are sharp, and one, assuming the form of an elevated curve, is more decidedly opaque or yellow than the others, and is known as the *arc of propagation*. Immediately behind it the ulcer with its gray floor seems deeper than the portion next to the corneal margin.

The surrounding cornea is opaque, and the lesion spreads rapidly, at the same time growing deeper; iritis, iridocyclitis, and hypopyon ensue, and perforation and extensive sloughing of the cornea are likely to occur. Usually the patient complains of severe brow-pain and the eye is intensely tender. Vision is reduced to mere light perception. In other cases, while the local lesion is severe, the subjective symptoms of inflammation are almost absent. Kipp called attention to certain types of infected ulcer from the margin of which straight, or nearly straight, lines diverge in all directions obliquely through the parenchyma of the deepest layers, their ends being connected by interme-

diate striæ. They may be due to folds in Descemet's membrane or to cell infiltration.

Hypopyon, to which reference has just been made, may complicate both small and large ulcers, and consists of a collection of pus in the anterior chamber, varying in extent from a mere line to a quantity which well-nigh fills the chamber.

This appears as a yellow mass at the bottom of the anterior chamber, and is bounded above by a horizontal margin. If the collection is fluid, its position will shift with movements of the head; if it is tenacious, no movement can be observed. The pus is caused by an aggregation of leukocytes derived from the vessels about the periphery of the cornea and from those in the inflamed ciliary body and iris, the endothelium of which is cast off. In other words, the pus in hypopyon does not come from the cornea. The changes which take place at the posterior surface of the cornea, that is, pus-corpuscles derived as already explained and which accumulate at a point corresponding to the ulcer, penetrate Descemet's membrane and invade the cornea, constitute an important factor in the perforation of the cornea which so frequently occurs (Fuchs). Sometimes Descemet's membrane is ruptured without perforation of the cornea, and the pus in the cornea and in the anterior chamber are in direct connection.

The combination of ulcer of the cornea and pus in the anterior chamber has received the name *hypopyon keratitis*, which generally is limited to the type described as infective or creeping ulcer. Hypopyon, however, also occurs in association with infected ulcers which are not serpiginous.

Causes of Infected or Sloughing Ulcers.—The investigations of Uhthoff and Axenfeld have demonstrated that—(1) Typical *serpiginous ulcer* of the cornea with hypopyon is nearly always caused by the *pneumoccocus* (Fränkel-Weichselbaum capsulated diplococcus); this micro-organism may frequently be found in these ulcers in almost pure cultures. These ulcers are also caused by the diplobacillus of Morax and Axenfeld (*diplobacillary ulcers*), the bacillus of Petit, the Bacillus subtilis (Zur Nedden), and the streptococcus. (2) Sloughing ulcers not typically serpiginous are caused by infection with *staphylococci*, *streptococci*, and by *mixed infection*. Occasionally pneumococci originate ulcers which are not characteristically creeping; they may be variously disposed in the center, periphery, and intermediate zones of the cornea. (3) In addition to the micro-organisms mentioned, the following bacteria, according to Uhthoff, have been found to be the cause of various forms of infected ulcer: Pfeiffer's capsulated bacillus, Bacillus pyogenes fœtidus, Bacterium coli, Bacillus pyocyaneus, diplobacillus, pneumobacillus, ozena bacillus, tubercle, and lepra bacillus. Other unidentified varieties have also been found, and corneal ulceration has also been ascribed to *streptothrix* (De Bernardinis).

Infected ulcer due to *Bacillus pyocyaneus*, as a rule, is a malignant process. Preceded by superficial keratitis a deep lesion develops, without the crescentic shape of the serpiginous ulcer and containing pus,

chiefly in its center. The diagnosis depends upon a bacteriologic examination.

The various micro-organisms come from the conjunctiva, the ciliary borders, the nares, the lacrimal passages, and from external contaminated surroundings. Infection of the lacrimal sac is responsible in fully one-third of the cases. An abrasion of the cornea caused by a chip of stone, a fragment of iron or steel, a chestnut-burr, beard of wheat, or twig of a bush is quite sufficient to make a port of entry for the micro-organisms, hence coal-miners, stonemasons, farmers, indeed, all workmen who are exposed to injuries of the corneal epithelium and whose conjunctiva or lacrimal sac is infected are peculiarly liable to this affection. Heat and hot weather, according to Fuchs, may be regarded as predisposing causes. Serpiginous ulcer may be caused by acute infectious diseases, especially smallpox (page 275). Typical manifestations of this disorder do not occur in children, but rarely they are attacked by a pneumococcal infection of the cornea which exhibits a notable severity in its manifestations, the ulcer being greatly infiltrated and the cornea liable to disintegration.

5. **Mycotic Keratitis** (*Keratomycosis Aspergillina*).—In a small percentage of cases of sloughing keratitis the infection is due to a mold—*Aspergillus fumigatus*—the fungus, as a rule, finding entrance through a corneal abrasion from injury. The ulcer has a dry appearance and has been compared to a grease-spot; it is surrounded by a gray or yellow line, and the enclosed area ultimately exfoliates. Hypopyon and iritis may be present; sometimes the lesions assume the form of a simple corneal infiltration resembling fascicular keratitis. Ellett has reported a corneal ulcer in which he found *Aspergillus nigricans*. According to Morax, mycotic corneal affections may be due to the *Verticillium graphii*.

6. **Abscess of the cornea** consists of a purulent infiltration in the deeper layers of this membrane, over the center of which, in the early stages, the epithelium is unbroken and prominent, but later discolored and slightly sunken.

The corneal zone immediately surrounding it is hazy. The margins of the collection are thicker and more prominent than its middle; pus is seen in the anterior chamber; the aqueous humor is turbid and the iris inflamed.

Generally the lesion grows more yellow, notches laterally, bulges forward, and finally bursts, leaving a more or less ragged ulcer covered with tenacious pus, and pursuing a course similar to that described under sloughing or infected ulcer, of which, indeed, abscess is the first stage. The causes are identical with those described in connection with infected ulcer; occasionally a definite cause cannot be ascertained.

7. **Infected Marginal Ulcer.**—According to Zur Nedden, this form of corneal disease consists of a 1 to 2 mm. long oval ulcer, running parallel to the limbus, with only a slight diffuse infiltration in its vicinity, the rest of the cornea being normal. Sometimes several superficial infiltrations develop, which may unite with the original ulcer and form

a sickle-shaped lesion; exceptionally the cornea is completely surrounded. In other cases the multiple infiltrations do not coalesce. The infection is believed to be due to a specific micro-organism, to which Zur Nedden gives the name "bacillus of infected marginal ulcer." The prognosis is good; hypopyon rarely forms.

Other types of *marginal ulcer* of the cornea are encountered to some of which reference has been made, for example, in association with catarrhal, diplobacillary, and acute contagious conjunctivitis. Marginal ulcers may arise as a complication of influenza. In a number of them, observed by the author, they began as grooved lesions just within the limbus, became turbid, and spread laterally. In two of them the severity of the process was so great that they resembled *rodent ulcers*. Influenza bacilli were not found, but either a pneumoccocus or a mixed infection.

8. **Exanthematous Keratitis.**—Most violent forms of *suppurative keratitis* occur during the convalescent stages of smallpox, though pustules rarely form upon the cornea. Abscess of the cornea occasionally accompanies scarlet fever, influenza, measles, typhoid fever, typhus fever, and pyemia, and in these cases has been regarded as metastatic, the pathogenic material having been conveyed through the blood. Schirmer's investigations indicate, however, that the so-called *variolar abscess of the cornea*, which has hitherto been considered an endogenous infection, arises by penetration of the virus from without.

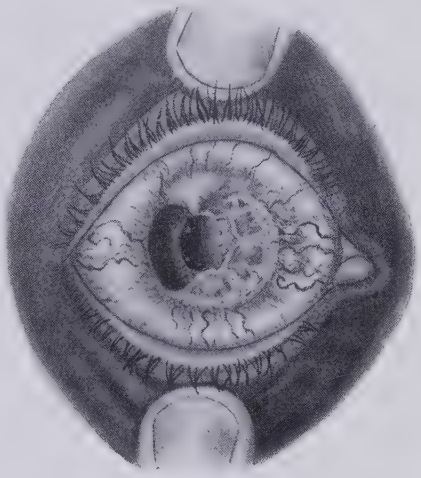


FIG. 127.—Mooren's ulcer (from a patient in the University Hospital).

9. **Ulcus rodens** (*chronic serpiginous ulcer, Mooren's ulcer*) is the name which was applied by Mooren to a *creeping ulcer* which begins usually at the upper edge of the cornea as a superficial lesion, separated from the healthy portion by a gray, opaque rim, which is undermined. The deeply undermined conjunctival edge of the ulcer is a striking feature of the disease. The extent of the undermining may reach 4 to 5 mm. from the border of the cornea. Mooren's ulcer may be associated with an ulcer of the sclera (Parsons). Although vessels may pass to a rodent ulcer and cicatrization apparently begin, it relapses quickly and progresses forward, until the whole cornea has been traversed and sight is destroyed. The cornea is not usually perforated in this disease, which is a rare form, sometimes bilateral, attacking adult and depressed subjects. The process may last from two to ten months and even longer. The cause of the disease has not been discovered, and investigations of its pathologic histology have failed to find an explanation of the limi-

tation of the lesion, at least almost always, to the superficial layers of the cornea (Feingold).

The *prognosis* is most unfavorable (see also page 280) in typical cases, but so-called *abortive* cases have been described, attributed to a neuropathic origin, in which the results of treatment are more satisfactory than in the ordinary types of this disease.

Fuchs has described *keratitis marginalis superficialis*, that is, a superficial variety of ulceration. which he has encountered in middle-aged persons, and which proceeds somewhat unevenly from the corneal border, so that the margin is indented toward the corneal center and framed in a gray line. The conjunctiva may be attached to it in the form of pseudopterygium. The lesion differs from ulcus rodens because it is more superficial and less undermined. It may last for long periods of time and is subject to relapse.

Ulcers in this situation in old people, the subjects of the uratic diatheses, may be quite small and may quickly appear, disappear, and reappear. Treatment along general lines is often of marked service.

10. **Circular ulcer** (*marginal ring ulcer, annular ulcer*) occurs in the form of a deep groove at the corneal margin, which gradually progresses until it may entirely girdle the cornea and cut it off from its nutrition. Photophobia, injection, lacrimation, and other irritative symptoms are not prominent, but perforation of the cornea and prolapse of the iris are common. The disease is seen in debilitated subjects. In the author's service in the Philadelphia General Hospital with its large contingent of enfeebled patients this type of ulcer was not infrequently encountered. Evidently, some of these lesions are analogous to, if not identical with, marginal degeneration of the cornea (page 308). In some cases, in addition to the ordinary local measures, covering the lesion with a conjunctival flap appeared to be of service.

Another variety of *ring ulcer* is formed as the result of a *marginal phlyctenular keratitis* (see page 267), probably by the coalescence of a number of small foci. Ring ulcers are also seen in catarrhal and purulent conjunctivitis, and in the latter condition may prove especially dangerous if they are hidden by the chemotic conjunctiva.

11. **Dendriform ulcers** (*keratitis dendritica; ulcerans mycotica; furrow-keratitis; kératite ulcéreuse en sillons étoiles*) are forms of keratitis which appear in branch-like ramifications, having a superficial situation, with slight knob-like swellings at the end of the branches. The cornea may be insensitive and fluorescein will stain not only the lesions, but the cornea exclusive of them (Verhoeff). The inflammation manifests itself in two forms.

In one, from the beginning, the symptoms include photophobia, lacrimation, strong bulbar injection, swelling of the upper lids, and absence of the epithelium over the furrow-formed ramifications—an implantation of the process in the deeper corneal layers.

In the other the disease assumes a subacute or torpid character, with practical absence of severe irritative symptoms and loss of the covering epithelium—a limitation of the lesion to the superficial layer. In

the first form the opacity is confined to the axis of the furrows; in the second, to the border. After healing, the scars have the same general configuration which was present during the stage of ulceration. The disease occurs in both sexes, and occasionally is seen in children.

The *cause* is not definitely known. The disease is attributed by C. J. Charles to a terminal nerve lesion, and by Verhoeff, who classifies it with neuropathic affections of the cornea, to disturbance of the nerve-supply. Many of the cases arise from febrile herpes of the cornea by an increase and coalescence of the small blebs. Indeed, the separation of dendritic keratitis in some of its manifestations from herpetic keratitis is largely artificial (page 294).

A keratitis in which the lesion consists of a peculiar, narrow, serpiginous, superficial ulcer, with lateral offshoots, like the skeleton of veins in a lanceolate leaf, usually associated with photophobia and lacrimation, and sometimes ushered in with severe supra-orbital neuralgia, has been attributed to malaria (*malarial keratitis*). It is a form of dendritic keratitis (Kipp) and has been well studied by Ellett.

12. **Exhaustion ulcer** (*keratomalacia*) may appear as an extensive ulceration in the center of the cornea, or as a ring abscess at its circumference. The tissue speedily is converted into a slough, which drops out, and an extensive perforation results.

In other instances the sequel is described as a species of *atrophy of the cornea*, which is converted into a whitish, flattened plate (Schmidt-Rimpler).

One or both corneæ may be affected, and the usual cause is exhaustion after acute illness or after prolonged diarrhea or dysentery. A similar softening and sloughing of the cornea may be the result of ophthalmia neonatorum (see page 217), or cataract incisions which have become septic, and xerotic keratitis (see page 291).

13. **Tuberculous Keratitis** (*Tuberculosis and Tuberculous Ulcer of the Cornea*).—Tuberculous lesions of the cornea almost always arise by reason of an extension of this disease from the uveal tract, including the pectinate ligament, and manifest themselves either as tuberculous nodules or as a diffuse parenchymatous keratitis (see also page 296). Primary tuberculous ulceration (Greeff) and tuberculous nodes in the corneal periphery (Bach) have been described which later push their way into the cornea. The lesion not uncommonly has a distinct triangular form (see Fig. 143). A variety of abscess of the cornea, without any healing tendency, which by some authorities has been regarded as a tuberculous process, has been observed in scrofulous children.

Prognosis of Ulcers of the Cornea.—This necessarily depends upon the character and situation of the corneal lesion, but even in the mildest forms some corneal opacity or irregularity of the corneal epithelium will remain (see page 286). If bacteriologic investigations should reveal the presence in the ulcerated area of pneumococci or of a mixed infection, the prognosis is serious, and at once the measures described in paragraph (*b*), page 280, should be instituted in the hope that the spread of infection may be prevented. In severe forms of suppurative keratitis the

prognosis is unfavorable, although active treatment is often followed by surprisingly good results; indeed, thanks to new methods of treatment, the prognosis is far better now than in former times.

Treatment of Ulcers of the Cornea.—It is not possible to lay down definite rules for the treatment of all forms of corneal ulceration—this must be governed by the exigencies of each case; but certain principles are common to the various types.

Acute Stage: Pain and Photophobia.—These should be relieved by the plans already suggested in the treatment of phlyctenular keratitis. In simple ulcers, atropin, a lotion of boric acid, and dark glasses will usually suffice, and prompt cure often follows an application directly to the ulcer of nitrate of silver (2 per cent.) or tincture of iodin.

Cocain will relieve photophobia temporarily, but its *continuous use in corneal ulceration is positively harmful.* Holocain, on the other hand, is of distinct value, as was pointed out by Hasket Derby, especially if applied directly to the ulcerated surface. If a corneal ulcer is accompanied by much dread of light, the methods described under phlyctenular keratitis may be employed. Dionin (2 to 5 per cent.) is of marked service.

Whenever corneal ulceration is associated with conjunctivitis, the inner surfaces of the lids may be brushed over with a solution of nitrate of silver, gr. ij–v to ʒj (0.13–0.32 gm. to 30 c.c.), or protargol (5 to 20 per cent.), or argyrol (10 to 25 per cent.) may be freely instilled. The last-named drug is not without danger, as it may cause a permanent brown stain at the seat of the ulcer. The culdesac should be carefully cleansed with a boric acid solution, physiologic salt solution, a collyrium of bichlorid of mercury (1 : 8000), or cyanid of mercury (1 : 2000), mercurophen (1 : 10,000) or mercurochrome (1 per cent.).

Subacute and Torpid Stage.—After the subsidence of the acute symptoms, or where the ulcer from the beginning is torpid, local stimulation should be secured with an ointment of the yellow oxid of mercury, gr. j to ʒj (0.065–3.885 gm.). Finely powdered calomel dusted into the eye is also of excellent repute. In like manner iodoform or aristol, in salve or powder, may be tried. Eserin has been recommended instead of atropin in small sluggish ulcers.

Deep and Sloughing Ulcers.—It was a universal and is still a common practice to instil a solution of *atropin*, because of its anodyne effect and because it lessens the liability to iritis. In the presence of active iritis or iridocyclitis the indications for its use are evident. The solution should be sterilized.

In some cases *eserin* (eserin sulphate or salicylate) is employed, because it stops the migration of white blood-corpuscles, or promotes absorption through dilatation of the ciliary vessels, or limits the sloughing process. Furthermore, abnormal intra-ocular tension is lowered by the action of the drug. The strength of the solution may be from ¼ to 1 grain (0.0162–0.065 gm.) to the ounce (30 c.c.), the latter being unnecessarily active in most cases. One or two drops of the eserin solution should be instilled from three to six times daily; and as, under its in-

fluence, congestion of the ciliary body and iris may ensue, as well as brow-pain, these complications should be counteracted by using a few drops of the atropin lotion at night. Deep ulcers near the margin are those most adapted to the eserin treatment. The author, after considerable experience, is persuaded that eserin in corneal ulceration, unattended with elevated ocular tension, has a comparatively limited value, and that atropin is usually the better drug.

During the progress of deep and serpiginous ulcers of the cornea a careful watch for *rise of intra-ocular tension* must be kept, and should it develop, mydriatic drugs should be discontinued; a myotic may be required or paracentesis of the anterior chamber. In certain deep ulcers situated at the margin of the cornea rise of intra-ocular tension and secondary glaucoma may readily manifest itself. Atropin, eserin, or pilocarpin may be used in conjunction with dionin, if this drug is indicated, to produce an analgesic or lymphagogue action.

Pain is relieved and the process of repair encouraged by the frequent application of *hot compresses* (see page 220), and by the use of dionin, which may be employed in solution or as an ointment. Hot water (150° F.) dropped directly upon the ulcer is recommended by Lippincott. The culdesac and lacrimal passages should be irrigated frequently with *antiseptic collyria*—a saturated solution of boric acid, bichlorid of mercury (1 : 10,000), aqua chlorini, cyanid of mercury (1 : 2000) or mercurophen (1 : 8000). Irrigations with a solution of sulphate of magnesium are recommended in sloughing ulcers (Kirkpatrick, see page 225).

1. *Impending Perforation.*—When a perforation of the cornea is imminent, a *dry antiseptic compressing bandage* should be applied which is removed when the necessary local applications are made, and again applied. Long-continued use of the bandage may be followed by eczema of the lids. This should be treated by dusting the parts with calomel or nosophen. Catarrh of the conjunctiva and dacryocystitis generally contraindicate the use of a bandage. If dacryocystitis persists in spite of ordinary treatment, the lacrimal sac should be excised (see page 794).

If bulging forward of the floor of the ulcer indicates that perforation threatens, the intra-ocular tension should be lessened by *paracentesis of the cornea.* This operation is described on page 723. It may be necessary to repeat the operation on several days. This procedure relieves pain and hastens healing.

2. *The Spread of Local Infection.*—If, in spite of such treatment, the corneal ulcer continues to spread, either in the form of a lesion creeping across the face of the cornea or by passing inward through its layers, the process must be stopped by one of several means: (1) Scraping with a curet; (2) the direct application of a suitable chemical which combines the properties of a germicide and a caustic; (3) thermotherapy; (4) the actual cautery.

(*a*) The ulcer may be cureted with a sharp spoon (under a boric acid spray—de Wecker), all the sloughed material removed, the edges penciled with a sublimate solution (1 : 2000), iodoform dusted upon its

surface, and a dry sterile bandage applied. Mules advised softened iodoform wafers.

(*b*) The chemical substances commonly employed are nitrate of silver, carbolic acid, nitric acid, trichloracetic acid, tincture of iodin, and formaldehyd. The first, in the strength of 10 to 20 grains (0.65–1.3 gm.) to the ounce (30 c.c.), is applied directly to the seat of ulceration (care being taken to avoid the surrounding cornea) by means of a probe on which has been twisted a thin band of absorbent cotton, or the point of a pencil of lunar caustic may be gently pressed against the sloughing tissue. Carbolic acid (liquid) may be employed in the same manner as the silver solution; or tincture of iodin, or a caustic solution of formaldehyd (1 : 50), or trichloracetic or nitric acid. Of these substances, carbolic and trichloracetic acid have given the author the greatest satisfaction. Absolute alcohol applied directly to the ulcerated surface is sometimes of great value. It should be used carefully and not too frequently, as it may produce bullous keratitis (Swanzy and Werner). This complication the author has never observed, and he has used alcohol frequently as an application to ulcers of the cornea. Prior to the application of these caustics the ulcerated area should be stained with a solution of fluorescein (see page 51). In place of iodin, concentrated Lugol's solution (iodin 25, potassium iodid 50, water 100) is recommended by Verhoeff in the treatment of hypopyon-keratitis. Prior to the application crucial incisions are made in the ulcerated area, followed by gentle cureting.

(*c*) The actual cautery may be either a small Paquelin or galvanocautery; when neither of these is at hand, a knitting-needle or platinum probe, heated red hot in the flame of a Bunsen burner, will suffice. The edge and floor of the ulcer should be gently but thoroughly burned. Usually one cauterization is sufficient, but in the event of failure to destroy all the infected material, the operation should be repeated on the following day (see also page 51). Cocain, holocain, or butyn render the operation painless, but there is no objection to general anesthesia in nervous patients.

If the surgeon is careful to touch only those portions involved in the ulcerated process outlined by means of fluorescein, it is said the resulting scar will not be greater than would have been the case had the ulcer secured cicatrization without such treatment.

Recent improvements in ocular therapeutics have rendered the use of the actual cautery less frequently necessary than in former times. In *rodent ulcer* (to which it should be applied early and thoroughly), however, it is one of the few means, according to Fuchs, that is efficacious, and it is also indicated in cases of fascicular keratitis. Swanzy and Werner highly recommend the application of absolute alcohol in Mooren's ulcer and record cures as the result of its influence. Siegrist advocates zinc-iontophoresis in this disease. He employs a solution of sulphate of zinc with a current of 2 milliamperes for two minutes. (See also footnotes, pp. 182 and 284.) Covering the ulcerated area with a conjunctival flap has been tried; this procedure the author employed in one case.

but without permanent good result. But F. A. C. Tyrrell reports one success with this method. The administration of thyroid extract has been tried in moderately developed cases with encouraging results (J. Herbert Fisher).

Abscess and Hypopyon.—The pus should be evacuated. If the ab, scess is unbroken, its anterior wall may be incised with a delicate knife- and the subsequent treatment conducted on the principles laid down for sloughing ulcers. If there is hypopyon, paracentesis of the cornea or the Guthrie-Saemisch section (see page 723) may be practised. Subsequently iodoform may be dusted upon the cornea and a bandage applied, to be renewed at suitable intervals. H. Gifford recommends *delimitating keratotomy* in the treatment of severe corneal ulcers. He makes a cut with a thin Graefe knife tangential to the ulcer and as close as possible to its most central edge, but without touching it. The wound is opened once or twice a day.

The antiseptic and specific treatment of ulcers have to a great degree replaced the operation of Guthrie-Saemisch, and in many instances absorption of the products of a hypopyon-keratitis follow non-operative measures.

Perforation.—If perforation of the cornea and prolapse of the iris occur, the vigorous use of atropin or eserin, according as the lesion has a central or peripheral situation, a compressing bandage, and rest in the recumbent posture represent measures which are sometimes followed by success. The advice occasionally given, that in these circumstances an effort to replace the prolapsed iris with a probe should be made, is not wise nor is the method feasible.

If the prolapse is a large one, the iris may be drawn forward through the aperture and excised close to the cornea. After excision the aperture may be covered with a conjunctival flap taken from the bulbar conjunctiva, twice as large as the original opening, into which it is gently inserted with a probe. A firm compressing bandage, not to be disturbed for three days, is applied. This is the method of Gamo Pinto. *Conjunctival flaps,* formed according to the technic of Kuhnt, are more satisfactory under these conditions than the Gamo Pinto operation. They are also useful in cases of rapidly advancing ulcer (see page 714). If the prolapse has been large, a more or less complete staphyloma is apt to follow in spite of vigorous bandaging and the use of eserin or atropin. An early *iridectomy* may prevent this catastrophe. In any event it is advisable to perform this operation as soon as it is safely possible after the healing process of an extensive ulcer has begun.

Other Methods of Treating Corneal Ulcers.—(*a*) *Dionin.*—The value of dionin in the treatment of corneal ulcer is unquestioned and has been referred to. It would seem that occasionally, in addition to its lymphagogue and analgesic action, it has a positive effect in stimulating corneal regeneration. Immunity is quickly established, and therefore, as a rule, it should be used for three days and then discontinued for two or three days, or until its application is again followed by the dionin reaction. The strength of the solution may vary from 1 to 10 per cent.,

according to the indications, a good average general strength being 5 per cent. Dionin may be combined with atropin, eserin, and holocain, according to the indications, but, in the experience of the author, furnishes better results if employed in a separate solution and the other drugs, also in separate solution, are used either before or after its application. Occasionally its action seems to be enhanced by the addition of adrenalin, although adrenalin itself is not a remedial agent of satisfaction. Powdered dionin or dionin in salve may also be used. With its employment by subconjunctival injection, as recommended by some surgeons, the author has no experience.

(*b*) *Serum Treatment.*—Römer, believing that 95 per cent. of infected so-called serpent ulcers are caused by the Fränkel-Weichselbaum diplococcus, with the aid of the chemist Merck developed a serum (*pneumococcus* or *antipneumococcus serum*) which he advises in the treatment of this form of corneal disease. The serum may be used subcutaneously and also subconjunctivally, and may be instilled into the conjunctival sac. Römer himself doubts the value of the subconjunctival injections. Subcutaneously from 3 to 5 c.c. of the serum may be employed, and the results thus far reported indicate that occasionally it seems to facilitate the cure of beginning ulcers, but in large, well-developed ulcers it is ineffectual (Zur Nedden). Complications have been reported—for example, myocarditis and decided febrile reaction are said to have followed the injections (Zeller). With this method of treating corneal ulcers the author has had no experience. According to Axenfeld, this serum possesses curative properties, but, as prepared at present, its action is not sufficiently certain to allow it to replace other methods. Recently Römer has advocated *autoserotherapy* in the treatment of hypopyon-keratitis. The serum obtained from a blister on the patient's arm is injected beneath the conjunctiva, the dose being 1 c.c.

The antistreptococcus serum or vaccine has also been employed in streptococcal infections, but apparently it, like the antipneumococcus serum, should be regarded as supplementary to other forms of treatment. Of staphylococcus serum-therapy too little is known to determine its influence on the eye (Axenfeld).

Antidiphtheritic serum has been utilized with success (Darier, Zimmermann, Fromaget, Key, the author) in the treatment of severe corneal ulceration and infection. Key, to whom we are especially indebted for investigating this remedy in this respect, points out that its advantage over other paraspecific agents consists in the fact that it is readily obtained, that its dosage is more certain and the preparation more dependable. The author has usually employed a dose of 1500 to 2000 units every other day for three days. His results have been remarkably good; it is a remedy of great value. Deutschmann's serum is a satisfactory agent (yeast serum in the dose of $\frac{1}{2}$ to $1\frac{1}{2}$ c.c. in children, and 4 to 8 c.c. in adults), according to reports by Deutschmann, von Hippel, and others. In Axenfeld's clinic its effects were negative. *Bacterins* prepared from the micro-organisms responsible for corneal ulceration have proved to be of value in the treatment of infected ulcer

of the cornea. The dose should be from 50,000,000 to 300,000,000. In the experience of the author they have acted favorably, but not more so than antidiphtheritic serum and other similar foreign proteins.

Hypopyon-keratitis has also been treated by means of applications of pyocyanase, prepared from cultures of the pyocyaneus bacillus.

(*c*) *Subconjunctival Injections.*—Naturally, subconjunctival injections, so satisfactory in certain diseases of the eye, have been tried in corneal ulcers. At one time bichlorid of mercury was chiefly employed, but, largely owing to Mellinger's investigations, this gave place to physiologic salt solution, which seemed to act equally well. There is a certain amount of evidence, however, that in infected corneal ulcers cyanid of mercury is the better agent. Of this drug, 10 to 20 minims (0.6–1.25 c.c.) of a 1 : 4000 solution may be injected beneath the conjunctiva; its use, in these circumstances, is strongly urged by Col. Henry Smith. There is no objection, but, on the whole, rather advantage in adding chlorid of sodium to the solution. Of the bichlorid of mercury solution, Dufour has recommended injections of 1 : 2000 if the ulceration is active, and 1 : 3000 to 1 : 10,000 if the infection is not very robust. Acoin added to the solutions employed in subconjunctival injections is said to diminish the pain. It should be used in a 1 per cent. solution, one-third of which is added to two-thirds of the solution employed.

(*d*) Since Weekers' advocacy of *thermotherapy* in the treatment of corneal ulcer this excellent method has been much employed. In this country Prince was the first to call attention to the great value of chauffage without cauterization—*pasteurization* as he termed it—whereby the ulcer is sterilized by means of heat, and to develop a satisfactory technic. The platinum blade of a galvanocautery may be used, or a small metal ball on the end of a rod heated to about 160° F. The lids being separated, the cautery is held close to, and moved slowly over, the surface of the ulcer (the ulcer must not be touched by it) for one minute. The surface of the cornea may be moistened with physiologic salt solution. This maneuver is repeated three times at each séance, and on subsequent days as may be required. W. E. Shahan[1] has designed a *thermophore*, an admirable instrument, with which the degree of heat can be exactly determined. For hypopyon-keratitis this should be 158° to 160° F. according to the character of the lesion. The applicator is brought in direct contact with the ulcer and kept there for one minute. The good results are usually not apparent until the second or third day.

(*e*) *Additional Measures.*—Reference to the value of iodoform directly dusted upon corneal ulcers has already been made. In its place xeroform or nosophen may be used. Sometimes these remedies are employed in the form of an ointment, as are also iodid of potassium, iodol, and europhen. The aniline dyes, in the form of blue and yellow

[1] The Thermophore in Ophthalmic Practice, Trans. Amer. Ophth. Soc., vol. xvi, 1918. This instrument has many uses other than those in the treatment of hypopyon-keratitis.

pyoktanin, at one time regarded with favor, in the opinion of the author are valueless. Iodin-vasogen is recommended in infiltrated and spreading ulcers in 0.6 per cent. solution (it may be applied directly to the ulcer with a cotton-tipped probe), and ariol dusted on the surface of an ulcer after the application of the actual cautery is commended by Fischer. If bacteriologic examination should reveal the presence of the diplobacillus in the corneal ulcer (*diplobacillary ulcer*), the preparations of zinc should be used (sulphate, chlorid, or salicylate in 1 to 5 per cent. solution) and applied directly to the lesion. Zinc preparations are also useful in pneumococcus ulcer, and Morax has tried rabbit's bile with indifferent success.

In recent years *ethylhydrocuprein* (optochin) has been strongly advocated in the treatment of pneumococcus ulcers of the cornea; it has also been used with advantage in ulcers not pneumococcic in origin (Holloway). It may be applied directly to the ulcer in a strength of from 1 to 2 per cent. by means of a cotton swab held in place for a minute and repeated as required, or used in a watery solution every hour, or employed in the form of an ointment, combined with atropin. Decided improvement has been noted as early as the third or fourth day, and even earlier. At first its application causes much burning pain, which may be checked with holocain. Some reports of its value are enthusiastic (Zentmayer); others express disappointment. The preparation should be freshly prepared and it is possible that failure to observe this precaution may account for some of these disappointments. Purulent keratitis has been successfully treated by zinc *iontophoresis*;[1] the application of light or *phototherapy* has also been used. Birsch-Hirschfeld recommends ultraviolet light in the treatment of ulcus serpens. Darier commends *scarlet red* in infected ulcers (5 per cent. ointment or an aqueous solution), and Haass urges *pellidol* and *azodolen*, derivatives from scarlet red, in 2 per cent. ointment in the management of corneal lesions with loss of epithelium. The application of radium has been advocated by Lawson and McKenzie.

The clinical use of *foreign protein* in eye infections has recently received considerable attention and the intravenous and intramuscular injection of *whole boiled milk* has been tried and, according to the reports, successfully in various ocular conditions, for instance, keratitis and corneal ulcers. Generally 3 to 5 c.c., or sometimes as high as 10 c.c., of the sterilized milk are injected into the gluteal or abdominal muscles. The author's experience with this remedial agent is too limited to express an opinion; in a few cases the method appeared, temporarily, to afford relief.

(*f*) *Associated Conditions.*—The treatment of conjunctivitis complicating ulcer of the cornea in nowise differs from that adapted to ordinary cases. An ulcer should always be carefully examined for the presence of a *foreign body*, which may be covered by a small slough,

[1] For the method of applying this remedial agent, see Traquair's article on "The Treatment of Purulent Keratitis by Zinc Iontophoresis," Ophthalmic Review, vol. xxx, 1911, p. 1.

while *misplaced cilia* are fruitful sources of corneal irritation and may hinder the prompt healing of ulcers. They should be removed with epilating forceps or destroyed by galvanopuncture.

The *lacrimal passages* should be explored and, if strictured, rendered patent, while irrigation of the lacrimal canal with a 4 per cent. solution of boric acid, or a 1 : 8000 solution of bichlorid of mercury, or a 1 : 10,000 solution of mercurophen, is of material aid in the treatment of infected ulcers, because this passage is commonly the seat of unhealthy secretion. If the tear-sac contains pus, it should be excised.

The *teeth* should always be examined, and, if faulty, the patient turned over to a competent dentist. The frequent relation of carious teeth and pyorrhea alveolaris to corneal ulceration is well established, and the irritation of a new dentition in young children has been found to be the cause of abscess or ulcer of the cornea. In brief, the entire *cephalic mucous membrane* should be explored, because, in one or other of its component parts, it may be the seat of disease, which, even if it is not the cause of the coexisting corneal ulceration, is none the less responsible for retardation in the healing process. Some corneal ulcers appear to have been caused by disease of the accessory sinuses, especially the ethmoid sinuses, and by purulent rhinitis, and if the tonsils are infected they should be removed.

Constitutional Treatment.—The patient, other things being equal, should not be secluded in a dark room, but, with eyes properly protected with colored glasses, go out into the fresh air every day. The diet must be nutritious and easily digested; tea, coffee, candies, and pastries should be forbidden.

If scrofulosis is present, cod-liver oil, lactophosphate of lime, and iodid of iron or syrup of hydriodic acid are indicated; anemia is best treated with the tincture of the chlorid of iron or with the carbonate of iron; any suspicion of malaria requires the use of quinin and arsenic. The syphilitic taint, which may be present without being the direct cause of the ulcer, indicates the iodids and mercury, especially in the form of the bichlorid. If ulcers of the cornea occur in gouty or so-called rheumatic subjects, citrate of lithium, mineral waters, iodids, colchicum, salicylic acid, salol, etc., are indicated. Thyroid extract has been used by the author and Veasey in stubborn non-ulcerative keratitis, and this drug has recently again been recommended by Radcliffe; it has also been tried in suppurative keratitis. In tuberculous ulceration of the cornea (see page 277) important results are achieved by the administration of tuberculin (see page 352).

A very strict inquiry into the condition of the alimentary canal should never be forgotten. Calomel is a useful laxative; the salines and saline waters are often necessary.

The urine should be carefully examined for albumin and sugar, and for the products which indicate imperfect assimilation. The influence of enterogenous auto-intoxication must be eliminated.

A very important element in the successful management of cases of sloughing ulcers, especially in subjects of depressed nutrition, is the

maintenance of proper circulation; strychnin and digitalis are often indicated. Severe pain may be alleviated by opium or morphin in suitable cases; the drug also has a favorable influence upon the ulceration. Codein also serves a useful purpose.

Results of Corneal Ulceration.—Opacities more or less permanent follow almost all ulcerations of the cornea. If the opacity is slight, it is spoken of as a *nebula* or *macula;* if dense, as a *leukoma.* An old corneal macula possesses a good reflecting surface, which serves to distinguish it, as Haab points out, from a recent inflammatory infiltration, which has a dull surface. Although usually white in color, maculas may be yellowish brown and contain dots or streaks of similar color. This condition is due to the deposition of hemosiderin and represents a form of degeneration.

It is evident that upon the position of the opacity in the cornea depends its influence upon vision. The more central it is, or, rather, the

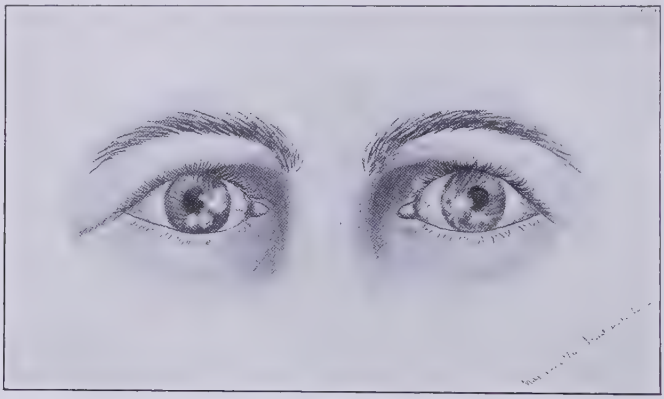


Fig. 128.—Adherent leukomas (from a patient in the Philadelphia General Hospital).

more directly it encroaches upon the pupillary region, the greater will be the disturbance of direct vision. Inequalities in the curvature of the cornea distort the retinal images and are fruitful sources of irregular astigmatism.

Where perforation has followed ulceration and the iris has remained entangled in the aperture, the attachment is called an *anterior synechia;* the corneal scar to which the iris is fastened receives the name *adherent leukoma* (Fig. 128). An eye thus afflicted may become quiet and retain, either with or without operative interference, useful vision; but may also be subject to recurring attacks of inflammation, and may cause sympathetic irritation or inflammation in the fellow eye. It may also become the subject of glaucoma.

The distention of a cicatrix, to whose inner surface the iris is attached, constitutes a *corneal staphyloma,* which is called *total* if the entire cornea is involved, *partial* if only a portion is included, and *racemose* if perforations have occurred at various points.

The mechanism of the development of staphyloma is, briefly, as follows: A perforation takes place, and the iris falls forward and attaches itself to the opening, or protrudes through it, becoming fixed there by the lymph thrown out in the process of repair. The scar tissue which

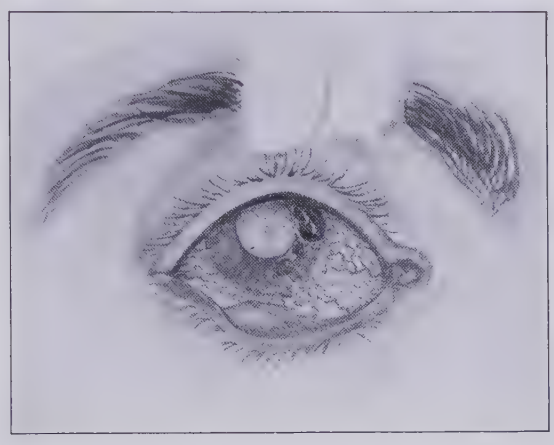


FIG. 129.—Beginning staphyloma following an infected ulcer which has perforated the cornea.

remains fails to withstand the intra-ocular pressure, and that portion of the cornea is pushed forward beyond its normal limits, forming a pouch-like deformity.

The protrusion may flatten down, and under the influence of fresh inflammation bulge forward again, or may extend between the palpebral

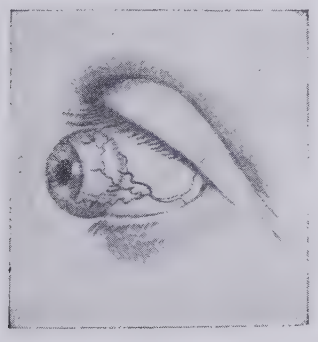


FIG. 130.—Complete staphyloma of the cornea.

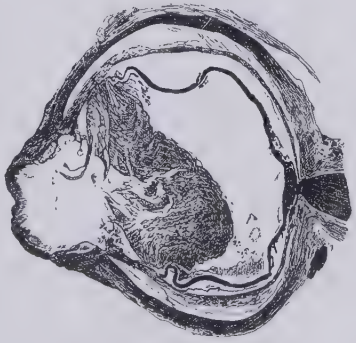


FIG. 131.—Section of an eyeball with complete staphyloma of the cornea; the lens has been removed.

fissures and prevent the lids from closing (consult Fig. 130). Staphylomas, the result of ulceration, are more or less opaque, because they represent the scar tissue which has formed after the rupture of the membrane. Corneal staphylomas, which are not opaque and have not

formed under the influence of an inflammation, also occur, and will presently be described. Thick corneal scars and staphylomas may undergo retrogressive metamorphosis with the deposition of hyaline masses and lime particles in them. Purulent ulcers may develop and perforate the lesion; they may even cause panophthalmitis and subsequent atrophy of the eyeball. Ulcers thus formed were named by Arlt *atheromatous ulcers*. The condition is also called *scar keratitis*, and, according to Fuchs, is due to entrance of bacteria through the diseased and feebly resisting epithelium (see also page 470).

Anterior synechia and adherent leukoma may also occur, according to von Hippel, from *internal ulcer*, without suppuration of the anterior corneal layers. Later the eye may become staphylomatous. The involvement of the cornea in pathologic alterations which come from behind has been elaborately investigated by Fuchs.[1] A purulent exudation, for example, in *endophthalmitis*, comes in contact with the posterior layer of the cornea and creates a purulent infiltration of that part of this tissue, that is, an *ulcus internum*.

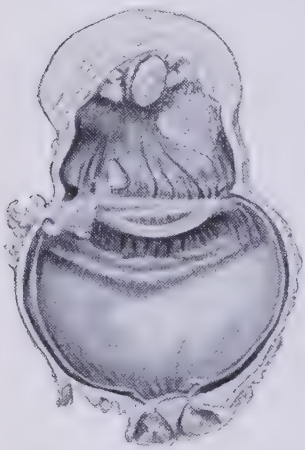


FIG. 132.—Staphyloma of cornea, with hypertrophy of the cicatricial tissue.

If after inflammation of the cornea, with loss of its superficial layers, the intra-ocular pressure causes the remaining lamina to bulge forward into an opaque elevation, the condition is called *kerectasia*. This differs from an ordinary partial staphyloma because there has been no perforation, and the iris tissue is not involved in the process.

If all the layers of the cornea down to the posterior elastic lamina are destroyed, and this protrudes through the opening in a small, translucent, hernia-like pouch, surrounded by a rim of opaque cornea, it is known as a *keratocele*.

An orifice remaining after a wound or, more commonly, because of the failure of an ulcer to heal is designated *fistula of the cornea*. It may last for a long period and stubbornly resist curative efforts. It has been recommended to touch the mouth of the fistula with a point of lunar caustic, and even to pare the edges and introduce a corneal suture. The best method of procedure is to cover the fistula with a conjunctival flap. Occasionally, the closure of a fistula of the cornea is followed by an acute rise of intra-ocular tension (glaucoma). A cicatrix of horny nature growing from the cornea has been reported by Arnold Lawson, and he thinks the epithelium covering cicatrices may not infrequently become cornified. An unusual result of chronic inflammation of the cornea consists in the development of *cholesterin crystals* (Greeves and Mayou) within that structure.

[1] Archiv. f. Ophthalmologie, 1916, Bd. 92.

Striate clearing of corneal opacities, usually in elderly persons, the scars having been present for a long time, noted by Nettleship and first described by Fuchs, consists in a metamorphosis of corneal cicatrices



FIG. 133.—Types of vessels in the cornea: *A*, Arborescent type; *B*, terminal loop type; *C*, brush or besom type; *D*, umbel type. (Courtesy of W. T. Holmes Spicer.)

in that they are divided in triangular and square areas of striæ, or a star-shaped figure is formed owing to a radial disposition of the clear

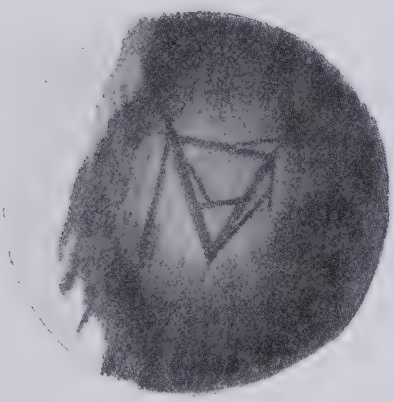


FIG. 134.—Clear geometric lines in a nebula. Terminal loop vessels at limbus. (Courtesy of W. T. Holmes Spicer.)

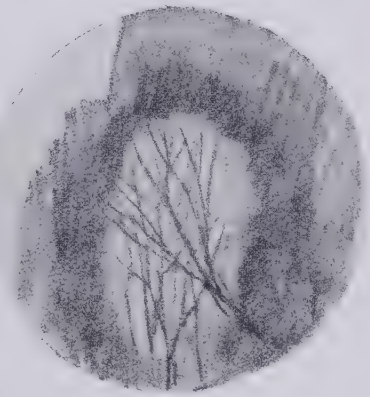


FIG. 135.—Interstitial vessels which afterward became geometric lines. (Courtesy of W. T. Holmes Spicer.)

lines. This condition has also been well described and illustrated by Sydney Stephenson and especially studied by Holmes Spicer.

The changes noted, according to Spicer, depend upon the presence

of blood-vessels. The types of vessels in cornea he has classified thus: (*a*) an *arborescent form* occurring in superficial inflammations, ulcers, or trachoma; (*b*) a *terminal loop* seen in marginal inflammations and ulcers and in vascular forms of interstitial keratitis; a *brush form* which is evident in interstitial keratitis and other varieties of deep inflammation; and an *umbel form*, which occurs in local inflammation of the deeper layers of the cornea. The study of the disposition of these vessels and that of the threads and clear lines which ultimately result is greatly facilitated by means of a Gullstrand diaphragm lamp and a corneal microscope. Veins and their accompanying arteries are usually distinguishable and the circulation of the blood can be seen.

The clear lines which have been mentioned Spicer describes as those which resemble *geometric figures* and those which suggest a *mushroom* cap or coral reef. According to him these caps are not scars of ulcers themselves, but secondary to them, and appear sometimes months after the ulcerative process has ended.[1]

Treatment of the Results of Corneal Ulceration.—Satisfactory results follow *massage of the cornea*. The massage movements should be made in a circular and radial manner over the cornea, through the closed lids, after the introduction of a small piece of the yellow oxid of mercury salve into the conjunctival culdesac. Some irritation accompanies the method, but may be allayed by the occasional use of a collyrium of boric acid and holocain. Instead of yellow oxid of mercury salve an ointment of dionin may be used, or the two remedies may be combined. In place of finger massage, *vibration massage*, introduced by Maklakow, may be employed. An Edison electric pen, the point being armed with a small ivory ball, is employed. The vibration rate varies from 200 to several thousand a minute. Subconjunctival injections of physiologic salt solution may aid in the absorption of corneal opacities following keratitis, and similar injections of magnesium sulphate and sodium sulphate have been recommended. Thiosinamin in $\frac{1}{2}$-grain (0.0324 gm.) doses and gradually increased has been employed in the treatment of opacities of the cornea. The author has tried the remedy without satisfactory results. Other methods of treating corneal scars are as follows: A 5 per cent. solution of a mixture of equal parts of thiosinamin and antipyrin (J. Galezowski); an eye-bath of ammonium chlorid, 1 to 3 teaspoonfuls to a cupful of boiled water (Pick); and injections of a 15 per cent. solution of fibrolysin, which is a compound of thiosinamin and salicylate of sodium, the dose being $\frac{1}{2}$ to 2 cm.

Alleman revived the use of *galvanism* for the removal of corneal scars, and reported favorable results. A suitably prepared electrode is connected with a battery, the cathode being applied directly to the anesthetized surface of the cornea, and the anode to the soft tissues of the cheek. Usually a current of from 1 to $1\frac{1}{4}$ milliamperes gives the best results. The *séance* lasts at the beginning of the treatment for one minute, and is gradually increased to three or four minutes. Great

[1] British Journal of Ophthalmology, 1919, 3, 1.

care should be taken not to produce too much reaction. Sulzer recommended *electrolysis* and *phototherapy* in the treatment of corneal opacities.

Vision may be improved by an iridectomy for new pupil, and the appearance of the eye by tattooing the cornea with India ink if the corneal leukoma is dense. Attempts have been made at transplantation of rabbit's cornea for the relief of dense central opacities. Some recent efforts at *corneal transplantation* have met with encouraging results, especially if a circle of clear cornea can be removed from a freshly enucleated human eye and transferred to the opening made in the leukomatous cornea. Von Hippel's trephine is a useful instrument for this operation. Magitot, employing *keratoplasty* and using very thin grafts, has obtained good results in the treatment of leukomas after burns, pterygia, and scars following trachoma.

The effect of treatment in *clearing* corneal opacities naturally varies according to the density of the lesions; that favorable results are secured by the methods suggested when they are not too dense (nebulas) is unquestioned. Although even a small corneal scar, for example, one remaining after a needle penetrates the corneal layers, may be permanent, corneal opacity arising in youth or in early childhood, for instance, after ophthalmia neonatorum, often in the course of time markedly diminishes.

The treatment of staphyloma in the first place is preventive, and those measures already described in connection with impending perforation of the cornea, and perforation after its establishment, are indicated—namely, a compressing bandage and the use of eserin or, in some circumstances, atropin. If, in spite of this, the bulging continues, paracentesis of the anterior chamber or an iridectomy opposite the clearest part of the cornea may be performed. Indeed, an iridectomy to prevent the formation of staphyloma is often useful while the corneal scar is still flat, especially if the tonometer indicates any rise of tension (see also page 281). If the disease has been so extensive that a complete and unsightly staphyloma has formed, which is the seat of pain and a source of danger to the fellow eye, excision of the globe is indicated, or one of the various substitutes for the operation of enucleation (see page 743).

In addition to the various types of corneal ulcer which have been described in the preceding paragraphs there remain other varieties of ulcerative keratitis more satisfactorily discussed in separate sections as follows:

Keratomalacia (*Xerotic Keratitis*—a name also applied to *keratitis e lagophthalmo—Necrosis Corneæ; Infantile Ulceration of the Cornea, with Xerosis of the Conjunctiva*).—This disease is characterized by dryness of the conjunctiva and destructive ulceration of the cornea, and usually appears in infants during the first year of life. According to Stephenson it is especially liable to arise about the eighth month, but may develop from the third to the twentieth months.

Cause.—Keratomalacia occurs only in anemic, badly nourished individuals. It has been seen accompanying meningitis, measles, and va-

riola, and among children with diarrhea, enteritis, tuberculosis, syphilis, and those who are inmates of homes whose surroundings are not hygienic. Bloch thinks a deficiency of fat in the food is responsible for many cases of infantile ulceration of the cornea. Bacilli have been found, but the special microbe, if it exists, has not been certainly isolated. In a few cases the Spirochæta pallida has been found (Stephenson). The disease is not a common one. A somewhat similar condition has been described in the eyes of negro children in the South (Kollock.) Experimenting on young rats by feeding them on a diet deficient in fat-soluble vitamin A, Yudkin and Lambert conclude that the keratitis which may develop in xerophthalmia (see also p. 254) does not begin in the cornea, but in the lids.

Symptoms.—In the beginning there are conjunctival congestion and lacrimation, but the peculiarity of the disorder is the development of the appearances described under Epithelial Xerosis (see page 254), in association with the corneal lesions. A gray haze, rapidly turning into ulceration, appears in the cornea, followed by inflammation of the iris and the formation of hypopyon. Perforation of the cornea and destruction of the eyeball may result. Both eyes, as a rule, are affected, one earlier than the other.

The **prognosis** is very unfavorable; the patients usually die (according to Stephenson the disease is fatal in 50 per cent. of the cases) of the wasting disease which has occasioned the trouble, or of an intercurrent pneumonia. In some cases streptococci have been found in the local lesions, and foci of these micrococci scattered throughout the body.

Treatment.—In addition to the ordinary treatment of severe corneal ulceration remedies which are indicated by the general state of the patient should be ordered. Bloch recommends the administration of cod-liver oil, and feeding the child with breast milk or whole sweet milk.

Neuroparalytic keratitis is the name applied to an ulceration of the cornea which arises because of paralysis of the trigeminus.

Causes.—Disease or lesion of the gasserian ganglion or of its branches or of the nuclei of the fifth pair, periostitis of the orbit, syphilitic deposits, and fracture of the skull may cause the affection. It frequently arises after removal of the gasserian ganglion for the relief of trifacial neuralgia, or division of the sensory root for the same purpose or the injection of alcohol into the ganglion or its branches. This keratitis, due to alcohol injections, may appear in a day or two or be delayed for a long period of time—a year or more.

The corneal lesion has been ascribed to a trophic change; to the lessened power of resistance which the cornea in its insensitive condition presents to external injuries; to irritation of the fifth nerve by the lesion; to micro-organisms; and to increased evaporation from the surface of the cornea.

Wilbrand and Saenger dismiss the traumatic theory and believe that some trigeminal fibers from the first branch remain and carry the irritation, which is finally concerned with the development of the disease. Such irritations, according to Verhoeff, might readily cause an

acid reaction, and if sufficiently long continued, originate the lesions. Hence, in the language of Parsons, the disease is probably due to irritative changes in and about the degenerating nerve.

Symptoms.—The keratitis preceded by slight dulness or cloudiness of the cornea begins in its center with a depression or exfoliation of the epithelium, and spreads peripherally until the central necrosis or slough separates, and perforation of the cornea with prolapse of the iris occurs. The anterior chamber may contain pus or pus mixed with blood. Beyond and around the central lesion the corneal tissue is comparatively clear, especially in a margin of 2 to 3 mm. in width, but in the periphery there may be secondary foci of infiltration, closely connected with inflammation of the neighboring conjunctiva. The surface of the cornea and conjunctiva is anesthetic. The intra-ocular tension is diminished. Pain and irritation are never conspicuous; indeed, these symptoms usually are entirely absent.

The **prognosis** in the absence of prompt treatment is unfavorable, and destructive inflammation often results, although occasionally the keratitis subsides without the formation of purulent material. The center of the cornea, however, is flattened and presents a dense scar at the termination of the disease.

Treatment.—The usual treatment of corneal ulcers is necessary; especially useful are dionin and holocain. The affected eye should be excluded from the influence of external irritants, either by a carefully applied antiseptic bandage or by a Buller shield, or, better, by stitching together the lids. If a *median tarsorrhaphy* is promptly performed the results are invariably good; indeed, the elaboration of the process is usually speedily checked. The lids should not be separated for a long period of time (weeks, even months) and after their separation the patient should wear protecting goggles. The same operation is indicated as a prophylactic measure preceding operation on the gasserian ganglion or its branches. Holocain persistently used prior to operation and the internal administration of sulphate of chromium are said to be of advantage. If the lids are not stitched together prior to operation, the eye should be covered with a protecting shield, such as has been devised by W. W. Keen. Experimental evidence indicates the propriety of preventing evaporation by keeping the eye in a moist atmosphere.

Keratitis e Lagophthalmo (*Keratitis of Desiccation; Keratitis Xerotica*—Feuer).—This affection arises because the cornea is exposed, owing to defective closure of the lids (see also pages 194 and 196). As the result of this exposure there is desiccation of the corneal epithelium, which becomes fissured and in places exfoliates. Thus, a pathway for microbic invasion is opened, and ulceration and suppuration occur. The usual *causes* of this affection are: proptosis of the eyeball, as in exophthalmic goiter (see page 670) and exophthalmos (see page 681); paralysis of the orbicularis, as in facial palsy (see page 194); and long-continued illness associated with defective closure of the lids.

The **treatment** consists in protecting the cornea by a suitable bandage, by a thin layer of vaselin (D. Smith), or by stitching the lids together (see page 696).

Herpetic Keratitis (*Herpes of the Cornea*).[1]—The corneal lesions associated with herpes zoster ophthalmicus have been described on page 177. The present disease consists of a vesicular eruption upon the cornea, which breaks down and forms an ulcer, characterized by a denudation of epithelium not unlike that produced by injury.

Causes.—Herpes of the cornea may be associated with whooping-cough, intermittent and typhoid fever, and, in general terms, with those affections in which herpes of the lips and nose is found. It is seen in acute and subacute disease of the posterior nares and pharynx, and also in infections of the respiratory apparatus generally (pneumonia, bronchitis), and may follow, or be associated with, influenza. In a number of instances it has followed *antityphoid inoculations*. The author observed during the late war a number of cases where this etiologic factor was in evidence, sometimes associated with herpes of the face. Lancaster refers to more than 20 cases under his observation, the primary lesion probably existing in the ganglia. During recent epidemics of influenza numbers of corneal affections developed, some of them of the herpetic type, which have been classified with the *neuropathic* group, where in addition to the corneal ulceration there were pain along the distribution of the branches of the trigeminus and anes-



Fig. 136.—Showing various shapes and positions of herpes-ulcers (Haab).

thesia of the cornea (see also Dendritic Keratitis, page 276). Febrile herpes, whether it appears on the face, lids (page 178), genitalia, or cornea, is not merely a symptom, but a specific infection. Recently Jonas Friedenwald, confirming and elaborating other observations, has made a complete demonstration of this fact by animal experimentation and the disease can be carried from one rabbit to the other. In addition to conjunctival congestion, punctate and dendritic corneal lesions appeared.

Symptoms.—The typical disease begins with the symptoms of catarrhal conjunctivitis followed by a series of transparent vesicles upon the cornea, which have been compared to a string of small beads. The vesicles are placed in a circle, or run in a diagonal or irregular line across the cornea. They speedily rupture and leave an open patch, deprived of epithelium, which is anesthetic and has irregularly serrated margins, upon which the remains of vesicles may be seen. The lesions are easily demonstrated by fluorescein, which may also produce a deep or superficial coloration of those portions of the cornea apparently unaffected. The sensation of the cornea is diminished.

[1] This term, as Horner observed, is often incorrectly used as synonymous with phlyctenular keratitis.

The progress of repair is slow, and is often interrupted by the reappearance of fresh vesicles. The herpetic ulcer may develop manifestations of great severity. Sometimes more than one herpetic ulcer appears at the same time, the intervening cornea being practically unaffected. A common disposition is a more or less central ulcer, with a second one crescentic in shape near the periphery. Occasionally the central lesion resembles a crudely depicted star. The disease may be associated with pus in the anterior chamber and iritis. Pain in the eye and brow, often violent photophobia, lacrimation, and a gritty sensation, are the subjective symptoms.

Treatment.—This consists in relieving the general condition; usually quinin in full doses is indicated, and salicylate of sodium is a most valuable remedy. Atropin, holocain, hot compresses, and dark glasses are needed. Dionin is of signal service. After the formation of the ulcer the treatment is conducted on general principles. A pressure bandage is of advantage, and in many cases an application of tincture of iodin is promptly successful. It may be repeated as often as required. Occasionally the application of carbolic or trichloracetic acid, thermotherapy, or even the actual cautery may be needed to subdue stubborn ulcers of this character.

Rosacea Keratitis.—Usually in women at or about the fortieth year of life, but sometimes at an earlier period in association with acne rosacea, a form of keratitis may develop. The manifestations vary. There may be lacrimation, blepharospasm, blepharitis, bulbar injection, and the development of a grayish-white, vascularized corneal infiltration with small circular ulcers, as in Holloway's patient; or small marginal ulcers and infiltrations may arise (Erdmann; see also page 207). The treatment of the coexisting acne rosacea is important. The eye lesions are favorably influenced by the usual lotions, by scopolamin-mydriasis, and especially by holocain.

Keratitis bullosa in many instances is a symptom and not a separate disease, inasmuch as it consists of the formation of one or more small blebs of short duration (*keratitis vesiculosa*), or of larger blebs of more enduring existence (*keratitis bullosa*), upon the cornea of an eye the subject of iridocyclitis, interstitial keratitis, or glaucoma.

Cause.—This affection formerly was attributed to a mechanical effect due to increased intra-ocular tension. Probably as the result of existing pathologic conditions an interepithelial edema takes place, the fluid penetrating from the anterior chamber through the changed and unresisting endothelial cells, or coming from the capillary network of the corneal limbus. This edema causes the epithelial cells to degenerate and loosen their hold on Bowman's membrane, and they are raised in the form of bullæ. Sometimes, in addition to epithelium, the walls of the bullæ are composed of a homogeneous membrane; or of a layer of new-formed connective tissue on the posterior surface of the bleb, which tissue is ultimately vascularized (*degenerative pannus*). Occasionally moderately large vesicles form upon a cornea otherwise normal, and in

one reported case malaria was believed to be the chief factor in their causation.

Symptoms.—In addition to the formation of the blebs, there are burning pain, photophobia, injection of the bulbar conjunctiva, and rupture of the vesicles, leaving an abrasion which may go on to ulceration, and its infection may produce sloughing of the cornea, and even panophthalmitis. There is a strong tendency to recurrence, and with each new formation of vesicles the violent inflammatory symptoms are repeated.

Treatment.—This consists in puncture of the blebs and suitable local measures, according to the causative disease. Holocain, dionin, and sometimes pilocarpin are useful. In severe cases iridectomy and even enucleation may be needed. The recurrent character and the remissions which have been described have suggested the use of antiperiodic doses of quinin; and these have been given with good results.

The second group of corneal inflammations, a description of which follows, is the *non-ulcerative*, and includes a variety of affections free from ulceration.

Vascular keratitis is a superficial vascularity (sometimes deep) and opacity of the cornea, and is seen in pannus caused by trachoma lids (see page 243), and in phlyctenular pannus the result of many relapses of phlyctenular keratitis (see page 268), and in certain types of parenchymatous keratitis (page 298). Such conditions, however, are also included in the general term *vascularization of the cornea*, and do indicate separate forms of disease.

A type of vascular keratitis characterized by the formation of two opposite vascular areas at the upper and lower margins of the cornea, which approach each other until the vascularization is complete, the intervening cornea being hazy or sometimes yellowish, resembling a purulent infiltration, was described by Carter. He believed that the disorder was of neuropathic origin, but evidently this condition should be classified with parenchymatous keratitis (see page 298).

Parenchymatous or Interstitial Keratitis (*Syphilitic, Inherited, Specific, and Diffuse Interstitial Keratitis; Anterior Uveitis*).—This is a diffuse keratitis in which a chronic inflammation of the whole thickness of the cornea takes place, until, almost always without ulceration, but always with superficial or deep vascularization, the cornea in severe cases passes into a condition of universal thick haziness.

Causes.—The majority of cases of interstitial keratitis are due to *inherited syphilis*, the evidence of which is present in at least 80 to 90 per cent. of them. In a small percentage of cases (2 to 10 per cent., according to Stephenson's investigations) *acquired syphilis* is the etiologic factor. Next to syphilis, tuberculosis furnishes the largest contingent of cases of interstitial keratitis (about 10 per cent., it is usually stated, a percentage almost certainly higher than the facts warrant). Forms of parenchymatous keratitis have been attributed to rachitis, malaria, myxedema, trypanosomiasis, leprosy, the climacteric, and depressed nutrition. Interstitial keratitis is occasionallly seen in an-

imals. It may be the result of trauma in an individual with hereditary syphilis. The relation of trauma to this disease has been noted by a number of observers (Spicer, Butler, Cunningham). Perhaps injury activates dormant spirochetes in the cornea (Hans Barkan).

The affection is most frequently observed between the ages of five and fifteen years (most frequent between six and twenty—Hoor), occasionally as early as the third year (exceptionally even earlier [Hutchinson, Cunningham]), but rarely after the thirtieth year. A few cases are on record as late as the sixtieth year of life. The disease is more frequent in females than in males, occurring in the former, it is usually stated, especially at the periods of second dentition and of puberty. Igersheimer's statistics, however, show no material difference in its incidence in the two sexes.

Parenchymatous keratitis appears to be aggravated by the development of menstruation, and also to undergo improvement at the time of the establishment of the menstrual molimen. It is probable that the affection occasionally arises *in utero*, and a *congenital form* of the disease, not differing in appearance from the ordinary or postnatal form, has been described (Randolph). It has been produced in animals by inoculation of the eye with syphilitic material, and in human beings it has followed a chancre on the lid or conjunctiva (10 cases, according to J. T. Carpenter). Spirochæta pallida have been found in the corneal layers. Fuchs has observed in some cases of interstitial keratitis that in shape the cornea is a vertical ellipse. In general terms he believes that an *oval cornea* is more frequent in persons with inherited syphilis and in those who are likely to acquire interstitial keratitis than it is in other patients. It does not follow, as he points out, that because the cornea is oval the patient has inherited syphilis.

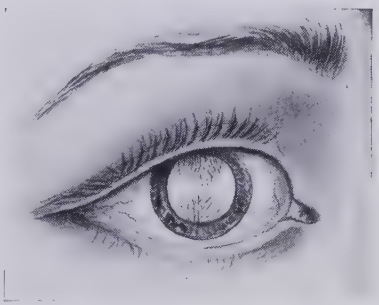


FIG. 137.—Vessel formation in the cornea after interstitial keratitis (see also page 289).

Symptoms.—The lesions begin either in the center or at the margin of the cornea. In the first instance, after a few days of slight ciliary congestion and watering, a faint cloudiness appears. The spots of haze, if carefully examined, will be found to be interstitial opacities, composed of round cells—that is, within the structure of the cornea itself and not on either surface.

In two or three weeks they spread until the whole cornea is invested with a diffuse haziness, veiling or completely hiding the iris, except, perhaps, through a narrow rim at the margin of the cornea. The steamy surface has often been compared to ground glass; it may have a yellowish tint. Careful inspection will reveal that the opacity is not uniform, but contains saturated whiter spots scattered through it, which have been described as "centers of the disease." Examina-

tion with the corneal microscope and slit-lamp demonstrates that the corneal haze may be resolved into very fine points of grayish color. Folds in Descemet's membrane of various shapes are also at times discoverable. A fibrinous reticulated exudation may be visible in the anterior chamber (Vogt). There are always at this stage ciliary congestion and some pain and dread of light. Blood-vessels derived from the ciliary vessels are thickly set in the layers of the cornea and produce a dull red color—"the salmon patch of Hutchinson." These patches may be small and crescent shaped, or large and sector-like. In one type (referred to on page 296), the vascularity creeps from above and below



FIG. 138.—The classical "map" of heredosyphilis, with all accessories, including bosses, corneal nebula, and Hutchinson incisors. Examination failed to reveal any evidence that this patient had ever had osteitis of the nasal septum to account for the flattened bridge. (Courtesy of Dr. J. H. Stokes, Mayo Clinic.)

until the entire cornea is cherry red. If the disease begins at the *margin* of the cornea, areas of cloudiness appear at different portions of it, and gradually from all sides approach the center until the general haziness is complete. Owing to the formation of vessels, the limbus becomes red and swollen at those portions which correspond to the marginal opacities, giving rise to an appearance which has received the name "epaulet-like swelling." It is most often seen in the upper corneal margin.

The subjective symptoms of irritability and photophobia are more pronounced in strumous children who are at the same time syphilitic.

Ulceration rarely occurs, but none the less ulcers of discoverable size are sometimes present, and hypopyon and an appearance resembling an accumulation of pus in the layers of the cornea have been reported. Iritis and iridocyclitis are not uncommon (fully one-half of the cases), in one form the iritis being associated with deposits on the posterior layer of the cornea (keratitis punctata, descemetitis) and the formation of anterior synechiæ; definite nodes in the iris are sometimes discoverable (Igersheimer). Severe inflammation of the ciliary region is occasionally encountered; secondary glaucoma and shrinking of the eyeball may follow (phthisis bulbi).

In the course of time, varying in accordance with the treatment, the cornea begins to clear, usually from the periphery. Perfect recovery of its transparency must be rare, although the remaining haze may be slight. Years after an attack of interstitial keratitis minute vessels, nearly straight, branching at acute angles and short bends, may be detected in the cornea. (See Fig. 137.) These appearances have been especially described by Nettleship and Hirschberg, the latter observer stating that the vessel formation never subsides entirely, and he has seen this condition, with the aid of a corneal loup, thirteen years after an attack. In Derby's investigation of the *end-results* of parenchymatous keratitis in some cases it was not possible to demonstrate the remains of vessel formation, although it is probable these observations were made without the aid of a slit lamp.

In addition to the *complication* of iritis and inflammation of the ciliary body, more or less retinitis is very apt to be present, although a pure retinitis is unusual. Disseminated choroiditis, and even optic neuritis and retinal hemorrhage, have also been observed; indeed, it is not uncommon to find, far forward in the eye-ground, areas of choroiditis (*anterior choroiditis*) not only in the diseased, but also in the unaffected eye (see also page 391). Secondary glaucoma may develop, with deep cupping of the disk. Hydrophthalmos has been reported (E. von Hippel). Periods of rise of tension during the course of parenchymatous keratitis are not uncommon, and must be carefully guarded against, by using eserin or by paracentesis of the cornea. It is important to make frequent tonometric examinations. Sometimes the tension is below normal. Myopia, sometimes of high degree and sometimes associated with glaucomatous cupping of the disk, and irregular astigmatism are not infrequent sequels of parenchymatous keratitis.

The subjects of typical forms of this disease often present a remarkable combination of physical defects. The dwarfed stature, the coarse, flabby skin, the sunken nasal bridge, the scars and lines at the angle of the mouth and also of the nose, the malformed permanent teeth, in which the central incisors have vertically notched edges (Hutchinson's teeth), indelibly stamp the inheritance of the patient. This character of teeth is present in between 20 and 30 per cent. of the cases. Indeed, it has been seen as frequently as 31 times in 48 cases. The presence of deafness, cicatrices in the pharynx, chronic periostitis of the tibia, synovitis of the knee-joint (symmetric or unilateral), and indurated

lymphatic glands further emphasize the syphilitic taint. Not only are the different forms of Hutchinson's teeth frequently evident, including the peg-shaped milk canine, but also the defective first permanent molars described by Fournier and Darier, and the "sloped molar" of Gifford.

The interstitial keratitis of *acquired syphilis* is usually a late secondary or a tertiary event. It may be circumscribed or diffuse, and is more apt to be unilateral than the variety due to inherited syphilis. Its evolution is relatively more rapid, and it is more promptly amenable to treatment. Usually it appears in adults between the twentieth and fiftieth year of life; exceptionally it has been seen in children (see also page 297). Its development as the result of a lid-chancre has been noted.

Diagnosis.—The course of the disease is usually quite typical, and the associated symptoms characteristic. The age of the patient in most instances helps to exclude primary glaucoma, while the history and character of the inflammation differentiate it from old corneal maculas and from the diffuse infiltration of the cornea which is sometimes seen as the result of injury. Wassermann's (or luetin) test should always be made, and, to distinguish between cases due to syphilis and tuberculosis, the reaction of the patient to tuberculin or to von Pirquet's test should be tried.

The presence of the minute straight vessels is good evidence of former parenchymatous keratitis. These vessels must be distinguished from those which remain after pannus from trachoma. According to Hirschberg, in the latter condition they are more superficial and pass into anterior conjunctival vessels. There are well-formed anastomoses, the broader veins are accompanied by finer arteries, and there are peculiar ramifications of the small deep vessels. The vessels seen in corneal scars after ulceration are confined to these cicatrices. The rest of the cornea is free.

Certain atypical cases of interstitial keratitis have been described, namely, forms in which the opacities are *stripe-like;* others in which they are *ring-like;* others presenting the appearance of pus in the layers of the cornea, the so-called *abscess forms*, with the central corneal area yellow in color and surrounded by intense vascularizations; others in which there is a combination of *parenchymatous keratitis* and *keratitis punctata*, and that form which is spoken of as *central annular interstitial keratitis*, especially described by Vossius, and usually seen in individuals under the age of twenty, and for which a definite cause has not been found. An *acute parenchymatous keratitis* associated with epidemic parotitis has been observed. The variety which begins as a *marginal vascular keratitis* has been described. It is difficult to distinguish precipitates in Descemet's membrane in this disease from dot-like lesions in the deeper layer of the cornea. Parenchymatous keratitis does not always begin primarily in the cornea. Iritis and iridocyclitis or rarely episcleritis may precede the corneal disease which then must be regarded as a secondary manifestation.

Prognosis.—From six to eighteen months are usually consumed in the development of the various stages of the disease. The second eye is almost certain to be attacked in from a few weeks to two months; even active medication may not prevent the involvement of the second eye. In rare instances the interval is many months, even a year; it may be delayed from five to six years. The patient or his friends must be warned of this fact.

A return to perfect transparency is unusual. The vessel formation in the cornea rarely subsides entirely, but even long-continued opacity in the course of time may markedly lessen, and reasonable vision be restored, especially if the refractive error (often myopia and astigmatism) is carefully corrected. The occasional onset of deep-seated inflammation of the ciliary region, and the fact that after the cornea has cleared evidences of choroiditis, retinitis, or disease of the optic disk and glaucomatous cupping, may be discovered, must not be forgotten in rendering a prognosis.

Relapses are frequent (18 to 22 per cent. of the cases, according to Hoor), not only of the corneal disease, but of the complications found in the iris and retina. Von Szili suggests that the relapses may be due to *anaphylaxis*, to which he also, in part, attributes the development of the disease after traumatism. It has been taught by some observers that the disorder is more severe now than in former times.

Pathology.—The principal changes occur in the deeper layers of the substantia propria of the cornea, and consist essentially of dense infiltrations of these areas. Newly formed blood-vessels are seen in the posterior and middle layers, and there may be nodular collections of lymphocytes (Fuchs). Some authorities distinguish between *primary* and *secondary* interstitial keratitis, the latter being associated with inflammation of the uveal tract. Leber regarded the disease as always secondary to a uveitis, and this view is strengthened, according to Parsons, by reason of the frequency with which anterior choroiditis can be found ophthalmoscopically in the less affected eye, and by microscopic examinations which have been made. Some authors (E. von Hippel, Elschnig, Stock) believe that this parenchymatous keratitis is primary—*i. e.*, that the corneal disease is the direct result of the general infection. Igersheimer is persuaded that this disease, at least in most cases, is independent of anterior uveal tract inflammation, and in so far as clinical observations are concerned should, as a rule, be considered as a specific parenchymatous disease of the cornea. Other observers (Stephenson) ascribe the corneal affection to an extension of the process from the uveal tract; that is, in syphilitic cases, the spirochetes, arriving from this area, proliferate in the corneal tissue. Referring especially to syphilitic parenchymatous keratitis, two views in regard to the nature are maintained, either that it is due to the direct action of the spirochetes which have been found in the cornea (E. von Hippel, Clausen, Igersheimer), or that it is an indirect manifestation of syphilis, *i. e.*, a para- or metasyphilitic affection. E. von Hippel, in some histologic investigations, found nodules in the cornea containing

epithelioid and giant-cells. Injections of *tuberculin T.* cause a local reaction in some of the cases, which suggests a tuberculous nature of the process.

Anaphylactic keratitis has been produced by Wessely, von Szili, and Arisawa which closely resembles human interstitial keratitis. Thus, Wessely obtained these results by injecting 2 drops of sterile horse-serum between the lamellæ of one cornea, followed in fourteen days by a similar injection between the lamellæ of the other cornea. Von Szili and Arisawa produced opacity and vascularization in a cornea sensitized fourteen days previously by means of an injection into the auricular vein. It is not improbable that interstitial keratitis should be regarded as an anaphylactic phenomenon. Thus, Derby, Walker, Igersheimer, and Schoenberg reason that the spirochetes or their toxins may sensitize the cornea during extra- or intra-uterine life. If later, during childhood, a new amount of latent syphilitic virus enters the previously sensitized cornea, keratitis results.

Treatment.—All irritating applications are harmful. Atropin, to maintain mydriasis, prevent iritis, and allay inflammation, should be systematically employed, unless rise of tension appears, when it must be discontinued. Dionin is of distinct service. Hot fomentations are useful, and tenderness in the ciliary region may be relieved by a leech applied to the temple in subjects of suitable age. The eyes may be protected from dust and light by smoked glasses or a dark shade.

A long-continued course of mercury is indicated. The most satisfactory method of administration in the earlier stages is by inunctions, 1 dram (3.885 gm.) of the ointment rubbed into the skin once or twice a day, according to circumstances. Mercury with chalk, 1 grain (0.065 gm.) three times a day, is highly recommended. Subconjunctival injections of bichlorid of mercury have been advocated, but in the author's experience have proved an unsatisfactory method of administering mercury in this disease. Similar injections of cyanid of mercury have been employed. Injections of saline solutions are in the opinion of some observers of decided advantage. L. Webster Fox advises subconjunctival injections of sodium saccharinate (1 to 3 per cent.). Some surgeons recommend that mercury be given in the form of hypodermic injections. An experience with this plan of treatment of this disease has not caused the author to abandon the usual methods of administration.

During the time the inunctions are being employed, cod-liver oil may be exhibited; later, bichlorid of mercury is a valuable remedy, and, as many of the patients are anemic, this is advantageously combined with the tincture of the chlorid of iron. Arsenic is useful, and atoxyl is highly recommended by Stephenson in doses of 0.25 to 0.50 gram, injected in the muscles of the back once or twice a week. A course of tonic treatment, nourishing diet, exercise, and healthful surroundings are necessary; in short, all measures are indicated which elevate the standard of the patient's general health. Indeed, it is most important to treat the subjects of this disease most carefully from the dietetic

standpoint. Injections of *tuberculin T.* in those cases depending upon tuberculosis have proved to be efficient. They should be administered according to the methods described on page 352. The subjects of parenchymatous keratitis, even though syphilitic, may have disturbances of the internal secretion. The administration of thyroid extract in some types of this disease possesses distinct advantage.

Much difference of opinion exists as to the value of *salvarsan* and *neosalvarsan* (or their equivalent, arsphenamin) in the treatment of interstitial keratitis of luetic origin. Uhthoff, for instance, maintains that this remedy does not favorably influence the disease. In the author's experience, on the contrary, this form of medication is most valuable, and in syphilitic patients it causes a subsidence of the irritative phenomena. Although it does not materially hasten the absorption of the corneal deposits, it definitely shortens the duration of the disease. The injections of neosalvarsan are given at intervals of one to two weeks; usually small doses are employed. During the intervals the medication should consist in mercurial inunctions, iodid of potassium, or mixed treatment. The dose of salvarsan must be regulated according to conditions and symptoms; usually each dose should be 0.4 gram. Sometimes 0.2 gram will suffice. A method of treating interstitial keratitis of syphilitic origin has been developed by Arnold Renshaw and J. Gray Clegg. A few drops of a 1 per cent. solution of *novarsenobillon* in distilled water are instilled at intervals of one week into the eyes; in late stages of the disease the interval should be fourteen days. The remedy should be used only as an adjuvant to the general treatment of this disease.

When all irritation has subsided, clearing of the remaining opacity is facilitated by the use of a salve of the yellow oxid of mercury, together with massage of the cornea, or by the local use of a solution of iodid of potassium. Subconjunctival saline injections may facilitate the absorption of the corneal opacities. Iridectomy, if the tension rises and glaucoma threatens, may be necessary; it is evident that it should be employed for new pupil if a stubborn central opacity remains.

Keratitis Punctata Syphilitica (*Keratitis Punctata Vera* [Mauthner]; *Keratitis Interstitialis Punctiformis Specifica* [Hock]; *Keratitis Punctata Profunda* [Fuchs]).—This form of keratitis was originally described by Mauthner, and is characterized by the appearance of circumscribed, pinhead-sized grayish spots in the parenchyma of the cornea; episcleral injection is usually wanting. The iris is not involved, the overlying cornea appears transparent, and the dots may arise quickly and disappear rapidly without leaving a trace. They probably indicate a *gummatous infiltration* of the cornea (more circumscribed gummatous infiltration or *gumma of the cornea* has been described). This disease is a rare manifestation of syphilis in its later stages, and should be treated with the usual antisyphilitic remedies.

Trypanosome Keratitis.—The ocular manifestations of trypanosomiasis in man have been studied by Morax, H. Lever, A. Laveran and Pettit, and a number of others, and include iritis, chorioretinitis, and parenchymatous keratitis. Trypanosome keratitis has been experi-

mentally produced (Stock and others). The author and Alan Woods used the *trypanosoma equiperdum* in their experiments and produced a typical parenchymatous keratitis—also iritis and retinitis. The corneal symptoms were always synchronous with the appearance of trypanosomes in the aqueous tumor. The ocular lesions yield (in animals) to repeated injections of salvarsan.

Keratitis punctata is characterized by a precipitate of opaque dots, often arranged in a triangular manner, upon the posterior elastic lamina of the cornea (Descemet's membrane—hence also called *descemetitis*). The overyling cornea is hazy, its surface at times slightly uneven. This affection is always secondary to disease of the iris, ciliary body, choroid, or vitreous, and hence is a symptom and not a special form of keratitis. It is fully considered elsewhere (see page 361).

Keratitis Profunda (*Central Parenchymatous Infiltration; Circumscribed Parenchymatous Keratitis*).—This form of keratitis is characterized by the formation of a grayish opacity in the deeper layers of the cornea, sometimes without severe irritative symptoms and unassociated with ulceration.

The *cause* is not always discoverable; sometimes alcoholic excess, cold, rheumatism, and malaria may originate the disorder; it undoubtedly may develop from an injury. W. T. Holmes Spicer believes that overeating and drinking and their results in the individuals or in their descendants, in the form of gout and rheumatism, with defective intestinal functions, are responsible for the majority of the cases. The disease is probably an expression of gastro-intestinal auto-intoxication in some cases. Influenza is a factor in the development of some of the varieties of keratitis profunda according to Hartman. An *acute interstitial keratitis* in association with mumps has been described, and deep infiltrates, therefore a form of keratitis profunda, may be one of the complications of herpes zoster ophthalmicus (page 177).

The following is Fuchs' description of this disease: The gray opacity, usually in the center, is covered by the superficial corneal layers, which are hazy and stippled, but not absorbed. Close examination (with a loupe) of the corneal opacity resolves this into individual points, spots, or gray interlacing stripes. The deposit slowly absorbs without ulceration, and commonly with only slight vesicle formation, and leaves the cornea clear, or permanent opacity may remain. Symptoms of inflammation may or may not be present; there is hyperemia of the iris. Slit-lamp investigation may reveal membranous exudation and cloudy flakes in the anterior chamber (Vogt). The duration of the disease is from one to twelve months, the average duration being about three months.

Spicer thus summarizes the symptoms of *deep keratitis:* Moderate ciliary congestion, moderate vascularization, but no salmon patch, opacification of the cornea, either as a central disk or a peripheral cone, an appearance under loupe examination of fine striated lines, and edema of the cornea. Fluorescein causes the deepest parts of the cornea to take on a stain. He believes that the true seat of the disease is in the nutrient blood-vessels.

The **treatment** requires atropin, dark glasses, and, later, yellow oxid or similar salve to aid resolution. Dionin and subconjunctival injections of salt or cyanid of mercury may be tried. The constitutional treatment is most important, and is governed by the probable cause.

Among the more uncommon forms of corneal inflammation the following may be mentioned:

Keratitis Superficialis Punctata (*Keratitis Subepithelialis Centralis; Keratitis Maculosa; Noduli Corneæ; Relapsing Herpes Corneæ*).—This disease appears in several forms, just as it has been described under several names, either different types of the same disorder or closely analogous manifestations.

Generally it begins with the symptoms of a sharp conjunctivitis; the secretion is watery, and there is catarrhal disease of the respiratory tract. In two or three days numerous small punctiform or linear spots appear, not immediately beneath the epithelium, but below Bowman's membrane. The overlying cornea is slightly hazy, and the epithelium above the spots a little elevated, the foci being more numerous near the center of the cornea than at the periphery. The cornea between the spots is somewhat hazy, and contains small points and gray lines radiating hither and thither, comparable to the fine fissures in ice. The disease is tedious and may last for months. Generally it occurs in young individuals, usually is bilateral, and is unaccompanied by loss of epithelium, ulcers, iritis, or hypopyon. In some cases, however, which at least begin with all the typical signs of superficial punctate keratitis, in the course of the disease there may be periods during which the fluorescein test will reveal many points which take in the stain. They do not seem to require a separate classification.

Stellwag described foci of large size, most commonly in the periphery. This type of the disease begins with pain in the brow, and the iris may be involved (*nummular keratitis*). It is analogous to interstitial forms of keratitis. In his cases the duration was much shorter, cure having been effected in two weeks.

Cause.—The anatomic nature of the spots is uncertain; by some observers they are regarded as enlarged and opaque corneal corpuscles or lymph-spaces filled with opaque matter. The disease may be associated with catarrhal and other affections of the upper air-passages and with infection of the tonsils. It has also been observed in association with menstrual disorders (Bosser), with recurrent fever (Trantas), and with influenza, but its exact nature is unknown. According to Verhoeff's investigations the affection is as a neuropathic keratitis, with the causal lesion in the ciliary ganglion. The infiltrations beneath Bowman's membrane he attributes to the action of pyogenic diffusible toxic substances arriving at nerve terminals. The disease differs from herpes in the absence of vesicle formation and herpes of the face, in its bilateral character, and in the great number of corneal spots or foci.

Treatment.—This should be directed to the mucous membrane of the nasopharynx as well as to the eye. If the tonsils are diseased they should be removed. Locally, during the state of irritation, atropin is

indicated, and later yellow oxid salve. Holocain and dionin are of service. Full doses of quinin are indicated, the salicylates and acetylsalicylic acid (aspirin) are valuable, and it has been recommended to use the constant current over the region of the distribution of the supra-orbital nerve.

Superficial Linear Keratitis.—This unusual disease, as described by Spicer and Greeves, generally observed in young adults, preceded by pain and congestion, consists in the formation of double contoured lines, raised above the surface of the cornea. Along these ridges are denser spots or nodes which sometimes stain with fluorescein. The lines are due to wrinkling of Bowman's membrane and the formation of new fibrous tissue in the adjacent substantia propria. Due to the folding of Descemet's membrane there is always marked lowering of the tension of the eyeball (hypotony). The lines may be vertical or nearly so and have slightly pointed ends; sometimes they cross each other roughly and resemble letters. The disease may run a mild course, or be severe and subject to relapse, in which case permanent opacities may result. It is possible this affection is akin to dendritic keratitis (page 276), but the disposition and form of the lesions are different.

Treatment.—The usual treatment of atropin, hot compresses, dionin, holocain, and applications of alcohol may be tried, but often the results are disappointing.

Haab has reported a form of keratitis in which lines, also double-contoured, cross and recross each other so that the appearance of the letters is produced, which he calls *alphabet keratitis* and which should probably be regarded as a process similar to, if not exactly like, the linear keratitis of Spicer and Greeves.

Keratitis Marginalis Profunda.—Under this name, Fuchs has described a rare form of keratitis in which a yellowish-gray zone of opacity, immediately joining the sclera, pushes into the clear cornea, accompanied by inflammatory symptoms, and occupies about one-half of the corneal circumference. The vessels of the limbus cover the opacity; in several weeks these and the inflammatory symptoms subside, leaving a rim of infiltration somewhat like an *arcus senilis*, save only that it joins the sclera directly and is not separated from it by a stripe of clear cornea. The disorder is unaccompanied by ulceration except in rare instances. It occurs generally in old people and usually is confined to one eye.

It should not be mistaken for the angular corneal opacity, which appears in connection with scleritis, and which is known as *sclerotizing keratitis* (see page 324); it differs from it in the absence of any preceding scleritis.

Keratitis Pustuliformis Profunda.—This unusual form of keratitis, described by Fuchs, is usually encountered in elderly persons and generally in men. Its lesions consist in variously placed and sized yellow deposits deep in the cornea and usually surrounded by grayish opacity similar to that seen in parenchymatous keratitis. Iritis is

always present and precipitates on Descemet's membrane, and hypopyon may be in association with the condition. The *cause* of the disease is not known; it appears to be syphilis in some cases. The course of the affection, one eye only usually being affected, is tedious. Resolution may take place, but generally permanent opacities remain and even flattening of the cornea. Evidently, according to Fuchs, a toxin derived from the inflamed iris attacks the cornea from behind. Treatment has proved to be of little avail.

Keratitis Disciformis (*Keratitis Annularis et Disciformis*).—According to Fuchs, this is an individual type of ring-like or disk-like keratitis. Verhoeff believes that, as it may be produced by a variety of causes, from the etiologic standpoint it is not a clinical entity. It should be distinguished from the annular keratitis of Vossius (see page 300). The disease is usually found in persons in middle life (in Weeks' statistics the youngest patient was eleven years of age and the oldest sixty-two) and appears frequently after slight epithelial defects, whether these are caused by injury or by herpes of the cornea. It is characterized by a delicate gray disk which occupies nearly the middle of the cornea, and which is separated from its transparent margin by an intensely gray, sharply marked border. The superficial layers of the cornea are smooth and unirritated. In the course of the disease, which lasts usually for several months, small ulcers may appear, and in most circumstances there is a decided opacity after the subsidence of the disease. With the aid of a slit lamp and the corneal microscope a loose coagulated exudation is at times visible in the anterior chamber (Vogt). Fuchs thinks that this disease has a position between serpiginous ulceration and the flat, disk-shaped ulceration after herpes of the cornea (see page 294). All three depend upon an infection of the cornea which gains entrance through a breach in the epithelium. The difference depends upon whether there is a deep or a superficial involvement of the tissue, which, in its turn, depends probably upon the character of the bacteria. Peters emphasizes the connection between this form of keratitis, corneal erosions, and serpent ulcer, and believes that all three depend upon a nervous lesion affecting the corneal epithelium, followed by edema of the tissue. Verhoeff contends that the disease is neuropathic in origin. Schirmer has described circumscribed parenchymatous keratitis, exactly resembling keratitis disciformis, due to infection by vaccine virus.

Treatment may include atropin, hot compresses, and the local application of absolute alcohol, but it has not been followed by encouraging success. Dionin should be tried. Weeks, finding tuberculin therapy valuable in the treatment of this disease, thinks it may be tuberculous in origin.

Grill-like Keratitis or Corneal Opacity (*Gittrige Keratitis*, Biber, Haab); **Nodular or Guttate Opacities of the Cornea** (Groenouw, Fuchs); **Family Punctate Degeneration of the Cornea** (Fehr).—Grill-like corneal opacity, known also under the name of *trellised and lattice-form opacity of the cornea*, was first described by Biber and Haab,

and has been well investigated by Freund. The last-named author gives to these opacities the following characteristics: They constitute a hereditary disease which appears first after the age of puberty, in the form of gray, superficially placed spots in and around the center of the cornea, which lie beneath the epithelium and lift it into a position of distinct unevenness on the superficial layers of the cornea, and by diffuse corneal opacity which, examined with a loupe, is seen to be composed of a grill-like network with radial opacities. The peripheral borders of the cornea are free from disease.

Nodular or *guttate opacities of the cornea* were first described by Groenouw and later investigated by Fuchs. According to these authors, the disease consists in the development of numerous small, rounded, or irregular gray opacities in the cornea, especially within the pupillary area. Between the larger opacities lie much smaller, dust-like gray points. The epithelium is slightly raised by the larger nodes, and, therefore, there is a certain slight irregularity of the corneal surface. Almost all of the cases have occurred in men, and they were not found to be associated with any constitutional disease. In some of the patients a history of previous corneal inflammation was obtained. *Reticular opacities* and *interstitial punctate opacities* are names also suggested for this disease.

With the name "family punctate degeneration of the cornea," Fehr has described a punctate opacity of the cornea which may affect several members of one family, and which begins about the tenth or twelfth year of life, progressing steadily until toward middle life. The cornea presents a diffuse gray appearance, and is strewn with white spots and dots of various shapes in the center, while the periphery remains comparatively clear. With a strong lens the diffuse opacity is seen to be composed of minute points, by the condensation of which the larger opacities are formed. The corneal surface is smooth, reflects evenly, and has normal sensibility. Although this corneal condition differs somewhat from the two previous ones just described, it evidently is analogous, as Fehr points out, to them, and he suggests that these lesions probably represent three different types of the same affection, for which he proposes the name "family punctate or spotted degeneration of the cornea."

These various processes represent a degeneration rather than an inflammation, and the opacities are probably due to deposits of hyaline material in the deeper layers of the corneal epithelium and in Bowman's membrane. A mucoid substance may be produced by degeneration of the corneal lamellæ. Nodular opacities of the cornea (Groenouw) are regarded by Wehrli as a form of chronic tuberculous disease of the anterior layers of the cornea (*lupus of the cornea*). Treatment is unavailing.

Marginal Degeneration of the Cornea (*Senile Marginal Atrophy*).—This is probably similar to, if not identical with, the *furrow-keratitis* of Schmidt-Rimpler, and has been well studied by Fuchs, George Coats, and a number of observers. It occurs in middle-aged

or elderly persons in whom usually an arcus senilis becomes wider and the cornea in the area of the arcus grows thin. A groove or gutter forms; this gives way before the intra-ocular tension, and a *marginal ectasia* develops. The symptoms of irritation are mild; sometimes the lesions are unilateral, sometimes bilateral. In Zentmayer's patient the grooves encircled the margin of the cornea of each eye except for an arc about 15 degrees down and in. Whether the process is degenerative or inflammatory has not been decided. Terrien has tried the application of the actual cautery for its relief.

Primary Progressive, Calcareous Degeneration of the Cornea.—Occasionally, as described by Axenfeld, corneas come under observation in which there has been a gradual development of a white calcareous, glittering ring, within which is a normal area corresponding roughly to the size of the pupil. This ring reaches to the temporal and nasal sides, close to the limbus, but is separated from it by a narrow, transparent band. Above and below it may not so nearly approach the limbus and may be less sharply defined. The vision through the central, unaffected area of the cornea may be quite good. The author has studied with the late Dr. Robin of New Orleans a very similar case, and has also seen one case in his own practice in an adult which began in comparative youth. Axenfeld excised a portion of the tissue for examination, and found highly refractile particles, soluble in acid; from this solution typical calcareous crystals were obtained. The epithelium was practically unaltered. Other progressive degenerations of the cornea are the chronic degenerations of a hyaline type, degenerations from the deposition of uric acid salts, and progressive fatty degenerations. (See also page 319.)

Epithelial Dystrophy of the Cornea.—According to Fuchs, this degenerative disease of the cornea affects only elderly persons, being more common in women than in men; it has also been noted by him in association with tabes dorsalis. The corneal sensibility diminishes, a diffuse opacity of the cornea develops in the pupillary area, associated with marked alterations in the epithelium, the surface of which is uneven and reveals blebs or small dark spots. While the epithelium is the chief site of the lesion, there is also stippling in the deeper layers of the cornea. There may or may not be increased intra-ocular tension. In two patients studied by the author the disease began in one, a man, eighteen years after a successful cataract extraction, and in the other, a healthy woman, without apparent cause. In a third case recently investigated added to the corneal changes there was marked rise of tension; eserin was of service. All the typical symptoms recorded by Fuchs were present. Treatment is said to be unavailing, but in one of the author's patients the persistent use of dionin, associated with the internal administration of biniodid of mercury, seemed to check the process. Duane recommends the administration of arsenic.

Filamentous Keratitis.—This somewhat unusual condition is characterized by the development of small threads or filaments of tissue on the cornea, which usually appear after abrasions or wounds (*trau-*

matic filamentous keratitis), or herpes, or occasionally without apparent cause (*spontaneous filamentous keratitis*). The tags have a bulbous extremity and are often twisted like a rope. They start from small vesicles by the formation of a slender pedicle, and are composed of epithelial cells, more or less degenerated, and sometimes especially elongated. Torsion of the filaments is due to the movement of the eyelids. A number of filaments may be found in a single cornea; thus, in a case reported by Zentmayer, fifteen to twenty of them in various stages, some as clear vesicles attached to the cornea by a short pedicle, others as filaments 5 mm. in length with a bulbous extremity, were noted. They may speedily disappear, or persist, or recur after removal. Locally, holocain is of service (see also page 777).

Riband-like keratitis (*primary opacity of the cornea; transverse calcareous band of the cornea; zonular opacity; keratitis trophica; keratitis petrificans* [Suker]) appears, as was pointed out by Nettleship, in two forms:

In the one, usually in elderly people, the exposed part of the cornea is invaded in a transverse direction by a *smooth subepithelial* opacity, oval in shape, which can be chipped off, and is composed of an incrustation of lime-salts. Hyaline deposits also appear in the cornea. There is no ulceration and no change in the overlying epithelium. The opacity is sharply limited and the remainder of the cornea is clear. The disorder almost invariably is symmetric, and is situated upon the exposed cornea, although deposits similar to the transverse band may also be found in other parts of the cornea. A margin of the cornea at each end is free. Gout and excess of uric acid in the blood have been suggested as constitutional causes, a suggestion strengthened by the occasional occurrence of insidious iritis, glaucoma, and hemorrhagic retinitis. It may be mistaken for the opacity which occurs from the injudicious use of salts of lead (*lead incrustation of cornea*). Deep-seated, pigmented, *band-like* opacity of the cornea has been described (Schründer), and the author has studied recently one example of typical bilateral zonular opacities, dark brown in color; no cause was found; the patient is an elderly woman.

In the other type of the affection a horizontal band of opacity, grayish brown in color, crosses the corneæ of eyes which have long been blind from iridocyclitis, sympathetic ophthalmia, and glaucoma. The stripe is less uniform and less sharply defined than the one previously described, and consists of a *roughened*, transverse opacity. The calcareous nature of the other type may be wanting. As it occurs in the lower third of the cornea, or that part exposed when the eye is rolled up, and in an eye with impaired nutrition, the affection has been considered dystrophic in its nature. According to Best, the deposits are composed of lime and connective tissue. Hyaline globules are often present.

Blood-staining of the Cornea.—This phenomenon has been observed in cases of hyphemia and increased intra-ocular tension and after injuries. It is not a frequent phenomenon (1 in 400 severe injuries [Römer]). The cornea assumes a smoky or rust-colored tint,

except at its periphery, the clear portion being sharply separated from the cloudy area, which, however, is usually more pronounced in its center. The appearances closely resemble those of an amber-colored lens dislocated into the anterior chamber. Bilateral blood-staining of the cornea following double iridectomies has been reported (H. Gradle). With the microscope numerous granules (probably hematoidin) are found deposited in the substantia propria, which, according to Griffith, have entered the corneal tissues by endosmosis in a state of solution. Koeppe's slit-lamp investigations indicate that both the interlamellar spaces and the lamellæ are affected, but the latter less so than the former. The lesions have been well studied by E. T. Collins, Vossius, Weeks, Verhoeff, and J. Griffith, and recently by Charles Maghy; according to the observations of Collins and the author, it requires at least two years for the stains to disappear.

Arcus senilis (*gerontoxon*), or a circle of fatty degeneration of the substantia propria just within the margin of the cornea, is, as its name implies, almost invariably found in old persons. A true arcus is always separated from the adjacent sclera by a thin stripe of clear cornea. Occasionally a genuine example of this affection appears to have been noted in children (Hansell). An arciform opacity, the result of ulceration, may be mistaken for arcus senilis. The nature of the fatty material in arcus senilis has not been determined (Parsons).

The affection requires no treatment, and its presence appears not to interfere with the healing of wounds; for example, in cataract extraction.

Senile degeneration in the form of *sclerosis* and *atrophy of the corneal margin*, which, according to Fuchs, may arise in connection with arcus senilis, has been described on page 308.



Fig. 139.—Conical cornea.

Corneal pits (dellen) have been investigated by Fuchs, usually at the temporal margin of the cornea. They exist in the form of small saucer-like depressions, and may arise in association with swelling, inflammatory or otherwise, of the neighboring conjunctiva, or spontaneously. They last from a few hours to several days.

Conical Cornea (*Keratoconus*).—This consists of a cone-shaped bulging forward of the cornea, and is rarely congenital. It has been observed in several members of the same family. Usually stated to be more common in women than in men, some recent statistics do not confirm this assertion. It occurs most frequently between the ages of fifteen and thirty years; rarely it is seen in children. Its exact cause is unknown. Exhausting illness, menstrual disturbance, and especially chronic dyspepsia may be associated with the development of conical cornea, the immediate cause being a disturbance in the relation of the intra-ocular pressure to the resistance of the cornea, that is, the intra-ocular tension is relatively too high. Minute clefts in Descemet's membrane may appear, and pulsation of the cornea synchronous with each

impulse of the heart. In the study of keratoconus von Hippel has applied Abderhalden's test and found a disturbance of glandular secretion, an observation which has also been made by Siegrist.

The cone is transparent in most instances; occasionally its apex is slightly opaque. The bulging slowly progresses, but does not rupture. After years it comes to a standstill. Ulceration by virtue of the increasing conicity of the cornea apparently does not occur, but an ulcer adjacent to the cone may develop as in a case of the author's where the small resulting cicatrix checked progress of the disease. One or both eyes may be involved, commonly the latter, the second eye being affected some time after its fellow. The eye usually becomes myopic; it is highly astigmatic; mixed astigmatism is commonly present. Slight forms of conical cornea may be overlooked unless the shadow-test is employed and the characteristic reflections observed, or a Placido disk is used (see page 51), or the cornea is studied with the ophthalmometer and the distortion of the images of the mires is investigated.

Treatment.—Although no form of glass or no optical apparatus may avail in advanced cases, a careful trial should always be made with spherocylindric lenses, and frequently their employment in unusual combinations will markedly improve visual acuteness. The refraction should be frequently investigated and the lenses changed according to existing conditions. The tendency for the cone to alter and increase in size seems to be lessened by the persistent use of eserin or pilocarpin. The strength of the solution need not be greater than $\frac{1}{12}$ to $\frac{1}{6}$ grain (0.0054–0.0108 gm.) to the ounce (30 c.c.). The administration of thyroid extract has been advised.

Properly perforated black disks—that is, forms of artificial iris—are recommended by L. Webster Fox for the relief of conical cornea.

If the apex of the cone appears to be thinning, a weak solution of sulphate of eserin and a compressing bandage are indicated. Indeed, the regular use of a bandage of this character at night, during the hours devoted to sleep, is of distinct value.

In advanced cases an operation is advisable, having for its object the substitution of a contracting cicatrix for the tissue at the apex of the cone, which shall diminish the excessive curvature. Several plans are suggested: (1) Cutting off a small, superficial flap and subsequently cauterizing the surface, associated with repeated paracentesis of the cornea, and later a small iridectomy for optical purposes; (2) cutting off the flap and drawing the edges of the wound together with delicate sutures; (3) cutting from the apex of the cone a small disk with a trephine; (4) multiple punctures with fine needles; (5) obtaining the desired loss of substance by the application of a galvanocautery. If the resulting scar is directly central, an iridectomy for optical purposes is usually necessary; but if the apex of the cone is eccentric, as it often is, iridectomy is not required. Elschnig advises that the galvanocautery shall be applied at a dull red heat to the apex of the cone, and this area connected with the nearest point of the corneal limbus by a superficially

cauterized band. Optical iridectomy, according to this author, is not indicated. (See also page 725.)

Injuries of the Cornea.—Traumatic Keratitis.—These comprise: (1) Foreign bodies, (2) erosions, (3) wounds, and (4) burns and scalds.

Foreign bodies, as particles of sand, cinders, fine splinters of iron, and bits of emery, may either lodge upon the epithelium or become embedded in the substance of the cornea. A splinter of iron, small thorn, or a spicule from a chestnut-burr may deeply penetrate the corneal layers or even pass through them.

The pain of a minute foreign body is considerable; the eye waters and grows red, and the source of irritation is commonly referred to the under surface of the upper lid, although the intruder may be directly upon the center of the cornea. A foreign body must not be wiped from its position with a probe or thin stick wound with dry cotton. Such a procedure almost invariably detaches an area of corneal epithelium and offers a port of entrance for microbic infection whereby an ulcer of the cornea may arise. The pernicious practice, only too common among workmen, of attempting to remove a foreign body by means of a tooth-pick or similar unclean instrument cannot be too strongly condemned; it often has been responsible for the development of a large infected ulcer of the cornea.

To remove an embedded foreign body a drop of a 4 per cent. solution of cocain or of a 2 per cent. solution of holocain or of butyn is instilled, the upper and lower lids are held apart with the thumb and forefinger of the surgeon's left hand, while with the right hand he takes a carefully sterilized needle or a spud, and lifts the body by a lever-like motion from its position with as little injury as possible to the cornea. The area should afterward be inspected by means of a cornea loupe and oblique illumination. In any case in which the operator is not sure that he has removed the foreign substance he may resort to the fluorescein method described on page 51. If the substance has been iron or emery, a small, rust-like spot will often remain. This should be removed, and, as this is frequently accomplished with difficulty by means of a spud, Sweet has designed a delicate instrument, constructed on the principle of a small drill, which is highly satisfactory. *Powder grains* may be removed by touching them with a fine galvanocautery point (E. Jackson).

If a long spicule has partially penetrated the cornea, its point being within the anterior chamber, it may be necessary to pass a broad needle through the cornea behind it to secure a surface against which to work, and to prevent the manipulations from pushing it entirely through the cornea and into the anterior chamber. Sometimes a spicule of metal in these circumstances is best removed by means of a magnet.

In the past war many cases of *multiple* foreign bodies were observed due to peppering of the cornea with fine metallic dust, the particles being so close together that the affected area resembled a cornea which had been tattooed with India ink. Under such conditions individual removal of the foreign bodies was out of the question. Sometimes the

bodies were gradually exfoliated and later those which remained were removed in the usual way. Fragments of unburnt *cordite* becoming embedded in the cornea and conjunctiva proved to be very troublesome and dangerous.

After the removal of the foreign body the resulting irritation may be allayed by a drop of atropin; the use of a bandage for a few days facilitates the healing of the abrasion. Disinfection of the conjunctival culdesac with a bichlorid lotion (1 : 8000) or one composed of cyanid of mercury (1 : 2000) is important. If in the attempt to remove the foreign body much abrasion of the cornea has occurred, great care should be exercised to prevent infection. Darier recommends a collyrium of cyanid of mercury which contains dionin.

Among oyster-shuckers a form of keratitis is prevalent (*oyster-shuckers' keratitis*), caused by small particles of oyster shells striking the cornea and producing ulcers. Randolph showed that the disease depends upon the irritating chemic ingredients in the shell, and not upon micro-organisms. It is best treated by atropin and mild antiseptic lotions, and a 2 per cent. solution of holocain in physiologic salt solution. Kerato-iritis, the result of a bee-sting, has been reported (Huwald), and also from the action of antipyrin (Inouye).

Erosions, Abrasions.—A superficial loss of epithelium caused by the contact of a sharp body, for example, a finger-nail, twig of a bush, beard of wheat, etc., in itself may be insignificant, but may lead, through infection, to a severe ulceration, particularly if the injured eye is exposed to the discharge from an inflamed lacrimonasal duct. The slight corneal changes which may be produced by repeated tonometric examinations have been referred to (page 95). Sometimes incautious manipulation of the instrument is responsible for a large abrasion of the epithelium; ulceration of the cornea may have a similar origin.

The **treatment** consists of the instillation of an antiseptic lotion, for example, bichlorid of mercury (1 : 8000), and the use of atropin and holocain, with a compressing bandage to immobilize the lids until healing takes place, provided no septic discharge is present. White's bichlorid-vaselin ointment is valuable (see page 762).

Relapsing Traumatic Keratitis Bullosa (*Relapsing Erosion of the Cornea; Traumatic Keratalgia*).—In general terms the symptoms of this affection are these: Some time—several weeks or several months—after an abrasion of the cornea by a finger-nail, a twig, or similar object, the patient experiences, almost always on awakening in the morning, some difficulty in opening the eye, followed, when the lid is raised, by marked foreign-body sensation, decided epiphora, flushing of the eyeball, and sharp neuralgic pain. Each movement of the lid is painful, and the "attack" continues from one-half to several hours, when, usually by afternoon, the symptoms subside and the eye is again apparently normal. Careful examination during the continuance of the irritative signs just described will reveal on the cornea a small ruptured vesicle, or a larger blister or bulla, or sometimes simply an erosion of the superficial epithelium, without indications of vesicle or bulla. Occasionally

the only lesion to be detected is the scar or macula caused by the original injury, without loss of epithelium. These attacks may recur at short or long intervals for weeks, months, and even years.

Treatment.—The ordinary treatment of corneal ulcer is indicated, and the author has been especially satisfied with the action of holocain (2 per cent.), persistently and frequently used. A pressure bandage and massage with a salve of yellow oxid of mercury may be tried. A drop of liquid vaselin instilled at bedtime is useful.

Wounds of the cornea naturally divide themselves into *non-penetrating* and *penetrating*, and differ in character according to the instrument which has inflicted them.

Non-penetrating wounds partake of the nature of abrasions, and, like them, may be in themselves of minor importance, but may result in sloughing ulcers.

The treatment already described is indicated.

A *penetrating* wound allows the escape of the aqueous; incarceration and prolapse of the iris may follow, with all the possibilities described in connection with perforating ulcers. The wound may injure the lens and cause traumatic cataract, or involve the ciliary region and cause sympathetic inflammation, or become infected and originate a sloughing keratitis or a panophthalmitis. Wounds of the corneoscleral junction are very common; usually they are associated with prolapse of the iris (*hernia of the iris*).

After a perforating wound of the cornea the eye should be thoroughly and promptly irrigated with bichlorid of mercury (1 : 8000) or cyanid of mercury (1 : 2000) and care must be taken to ascertain whether a foreign body has lodged within the anterior chamber or within the deeper portion of the globe. *x*-Ray examination may be necessary. The iris, if prolapsed, and if replacement is not possible, and usually it is not advisable to attempt it, should be seized with iris forceps and excised. Unless the coaptation of the corneal wound is absolutely perfect, it should be covered by a conjunctival flap.

Gaping central wounds of the cornea, according to de Wecker's method, may be covered with conjunctiva, which is dissected loose in such a manner that it may be united over the cornea by a purse-string suture. After the corneal wound has united, the conjunctival covering is removed and restored to its original position. Kuhnt's conjunctival flaps may also be used and usually more satisfactorily (page 714). The tendency to *traumatic iritis* may be combated by the frequent use of ice-cold compresses, and the instillation of an atropin solution, especially if the wound passes through *corneoscleral junction* and injures the ciliary body. Mercury should be administered; also salicylate of sodium. In severe corneal wounds, involving the iris, lens, and ciliary body, the question of enucleation or evisceration must be decided.

Burns and **scalds** are produced by the contact of acids, lime, molten metal, and hot water or steam, and the general management of such cases does not differ from that of similar injuries of the conjunctiva, which necessarily is involved (see page 264).

Sometimes the burn may be superficial and the whole surface epithelium may be changed into a white scum, which presents a most alarming appearance. The destroyed tissue, however, is speedily replaced by a new layer of epithelium. Burns with slaking lime and molten metal are liable to result in disastrous consequences, and may be followed by sloughing keratitis and even panophthalmitis, and if the burns are located at the limbus, elevation and intra-ocular pressure may develop (Kümmell). Allport and Rochester have advantageously used intramuscular injections of cacodylate of sodium in the treatment of corneal opacity following a lime burn. *Ammonia burns* of the eye are of serious import, and even where the injury originally seems to be comparatively slight, there may develop later rapid necrosis of the cornea, with exudation in the anterior chamber, followed by blindness. There is a certain similarity between *carbolic acid burns* and those caused by ammonia, but the ultimate prognosis is, according to Stieren, less gloomy.

Severe corneal and conjunctival burns have been caused by *exploding golf-balls*, so-called "zodiac" and "water-core" balls, containing caustic contents, *e. g.*, barium sulphate, sodium hydrate, soap, free alkali, and zinc chlorid. The *treatment* of burns and scalds has already been described (see page 264).

All the various forms of corneal injury cause more or less severe inflammation, properly classed under the general term *traumatic keratitis*, and possess in greater or less degree the cardinal symptoms of keratitis—pain, lacrimation, photophobia, and disturbance of vision (see also page 313).

Peripheral Annular Infiltration of the Cornea (*Ring Abscess of the Cornea*).—This condition is characterized by an infiltration of the cornea, the exudation being distributed in a zone concentric with the corneal margin. At first the ring is gray, but rapidly becomes yellow, its inner edges being somewhat less well defined than its outer; in almost all cases panophthalmitis is the ultimate result. The condition most commonly follows perforating wounds of the cornea, especially if caused by chips of metal, and by operations, for example, cataract extraction. Rarely it is seen after perforating corneal ulcers and in metastatic ophthalmitis. It has been well studied by Fuchs and by Morax. Bacteria enter the anterior chamber through a wound and there proliferate, and by their products give rise to an infected iridocyclitis and keratitis; the cornea is attacked from the rear and the condition represents its reaction to the toxins acting upon its posterior layers. Leukocytic infiltration in the form of a ring follows, which is itself amicrobic. Hanke believes he has found the specific bacillus, but Morax maintains that so-called ring abscess cannot be explained by the presence of any one specially determined microbe, but by the proliferation of certain microbes in the anterior chamber, among which he is willing to admit Hanke's bacillus.

Traumatic Striped Keratitis (*Keratitis Striata*).—This condition may arise after incised wounds of the cornea, and in its most perfect manifestation, after cataract extraction (see page 777), and especially

after expression of the lens in its capsule (see page 770). The gray striæ, which should be studied with a loupe or slit lamp, are disposed perpendicularly to the wound, and stretch toward the opposite margin of the cornea. Often they cause little or no irritation, and the appearance does not materially complicate the treatment of the wound which gives rise to them. These stripes do not represent a cellular infiltration, but depend upon folds in Descemet's membrane. Usually they disappear within a week of their development.

Obstetric Injuries of the Cornea (*Birth Injuries*).—These injuries may be due to prolonged labor or to forceps pressure. They have been particularly well studied by Ernest Thomson and Leslie Buchanan, who classify them as diffuse temporary opacities due to edema, and permanent linear opacities, which extend vertically, obliquely, or horizontally across a whole or a part of the cornea, and which are caused by rupture of the posterior elastic lamina of the cornea. In several cases of the edematous variety studied by the author the opacity gradually but entirely disappeared. Doubtless some of the scars of the cornea, known as "congenital leukomas," have been caused by birth injuries. The author has investigated a few cases of high unilateral irregular corneal astigmatism evidently due to the same cause.

Birth injuries of the eye are not confined to the cornea. Thus a rare condition noted by W. S. Franklin and Warren D. Horner consisted of subconjunctival swellings which followed trauma during labor and which depended upon an extension of orbital fat through Tenon's capsule.

Tumors and Cysts of the Cornea.—Tumors of the cornea are rare. The following have been described: fibroma, papilloma, myxoma, dermoids, sarcoma, epithelioma, and endothelioma. True fibromas have been reported, but usually they are *scar-fibromas*, that is, they are hyperplastic scars. A myxomatous degeneration of such a scar is the probable origin of the so-called corneal myxomas. The papillomas, which have been examined, in most instances have arisen at the limbus and invaded the cornea. A few primary sarcomas, even in children, have been described, and Parsons has investigated one endothelioma. According to this authority, nearly all of the cases of so-called epithelioma of the cornea are growths beginning in the limbus at the position where the conjunctival changes into corneal epithelium. This was the condition of affairs in the specimens examined by the author.

Dermoid tumor is a congenital growth, and sometimes is associated with other anomalies of the lid and eyes. Strictly limited to the cornea, it is most uncommon; generally it occurs as a firm, hemispheric, yellowish-white growth, lying partly upon the cornea and partly upon the conjunctiva. The apex, often paler than the rest of the growth, contains short hairs. These, however, occasionally grow to an unusual length, and have been seen protruding through the fissure of the lids and hanging down upon the cheeks. If undisturbed, the tumor may slowly enlarge, and has been reported to have attained the size of a walnut. Bilateral dermoids have been recorded.

These dermoids have been ascribed by Van Duyse to the remains of amniotic adhesions, and by Remak to invagination of the ectoderm. Microscopically, the growth represents the structure of the skin and its appendages.

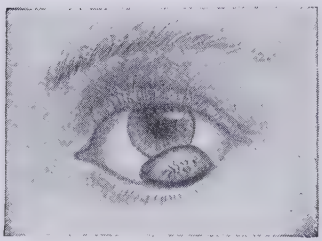


FIG. 140.—Dermoid of the cornea (from a patient in the Philadelphia General Hospital).

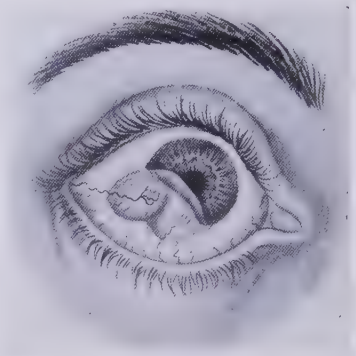


FIG. 141.—Dermoid of the corneoscleral junction, which on section showed a gland inclusion (from a patient in the University Hospital).

Teratoid tumors may be situated at the corneoscleral junction, rarely, if ever, on the cornea, or upon the outer half of the sclera, or upon the bulbus in the neighborhood of the caruncle. They contain, as a rule, acinotubular glands, fatty tissue, smooth and striated muscle-fiber, hyaline bodies, and cartilage.



FIG. 142.—Microscopic section of dermoid of corneoscleral junction with gland inclusion.

Corneal cysts, according to Oatman, occur in two principal forms—*epithelial* and *lymphatic*. The first variety is the more common; it usually arises from the epithelial layer of the conjunctiva, which is ingrafted on the cornea. Cysts following injury of the cornea have

been ascribed to the proliferation of surface epithelium which has been carried into the corneal stroma. They are, therefore, *implantation cysts*. Oatman doubted this pathogenesis, and explained them by assuming that a proliferation of superficial epithelium lines the wound with epithelial cells, and the mass thus produced, separated from connections with the surface, takes on an active growth and forms the cyst. *True lymphatic retention cysts* may result from dilatation of the corneal canals and spaces. Corneal cysts may be cured by excising a piece of their walls.

Congenital Anomalies of the Cornea.—*Microphthalmos* is that condition in which the entire eye remains in a more or less rudimentary state, and in which the cornea is too small in all its diameters. Pure cases of microphthalmos, according to Manz, are very rare; usually one or other of the component portions of the globe is wanting. Numerous theories have been advanced to explain their etiology—incomplete closure of the fetal ocular cleft (Arlt), fetal illness *in orbita* (Wedl and Boch), intra-uterine sclerochorioretinitis (Deutschmann). The affection has also been ascribed to the influence of heredity.

Megalophthalmos is described on page 442.

Sclerophthalmia or *sclerosis* is that condition in which the opacity of the sclera encroaches upon the cornea in such a manner that only the central portion remains transparent. It is due to an imperfect differentiation of the cornea and sclera at an early period of fetal life. It may by symmetric, and affect only the upper half of the cornea.

Congenital opacities of the cornea are seen in the form of milky spots which may clear up in later life, or as leukomas. Usually the iris is dimly visible through the clouded tissue. These opacities are due either to intra-uterine inflammation or to an arrest of development (see also Birth Injuries, page 317). *Embryotoxon* (*arcus juvenalis*) is a congenital opacity of the cornea which resembles an arcus senilis.

Congenital anterior staphyloma of the cornea appears in the form of a true staphyloma, and is a rare affection. The abnormality depends not so much upon a malformation or an arrest of development as upon a fetal inflammation, which, according to Pincus, takes place in the second half of fetal life. They have been well studied by J. Herbert Parsons, who holds that the lesions develop in exactly the same manner as they do when they take place after birth. Treacher Collins, however, thinks failure of the development of the anterior chamber may be the original cause in some cases. Peters ascribes the condition to defective development of Descemet's membrane, and E. von Hippel to *internal ulcer of the cornea* (see also page 288). Heredity probably plays some rôle in this and similar affections of the cornea. Congenital staphyloma of the cornea associated with dermoid formation has been reported.

Congenital melanosis or *pigmentation of the cornea* may appear in the form of a vertically oval area of brownish color in the center of this membrane. The affection is more common in women than in men. It has been ascribed to an abnormal development of the uveal tract

(Krukenberg). The lesion has been well studied in this country by T. B. Holloway. Congenital familial flatness of the cornea (*cornea plana*), according to Ruel, is characterized by flattening of the anterior portion of the eyeball in such a manner that the curvature of the cornea passes directly into the curve of the sclera without the formation of an angle. In Ruel's patients the corneas were diffusely opaque.

Pigmentation of the cornea, *not congenital,* has been noted. Thus Holloway has described *peripheral pigmentation of the cornea* in association with symptoms suggesting multiple sclerosis; some of the patients have cirrhosis of the liver.

Pigmentation of the corneal limbus has been observed in Addison's disease (Meesman). Examination of the deposit by means of the slit lamp demonstrates that the pigment is situated in the deeper layers of the epithelium. An unusual result of argyrosis is a somewhat grayish staining of the cornea (*argyrosis of the cornea*).

CHAPTER VIII

DISEASES OF THE SCLERA

The sclera, constituting four-fifths of the covering of the globe of the eye, and being in intimate relationship with the choroid and ciliary body, is subject to inflammations peculiar to itself, and to changes indicative of disease of these subjacent structures. Its close connection with the cornea associates this structure with some phases of its diseases, and its union with the iris through the pectinate ligament establishes an anatomic connection, just as there often is a pathologic relation. The overlying bulbar conjunctiva necessarily participates in scleral inflammation.

The inflammations affect (1) the episcleral tissue (*episcleritis*) and (2) the sclera itself (*scleritis*), and hence are *superficial* or *deep*. They further are *acute* or *chronic*, *diffuse* or *circumscribed*.

Episcleritis occurs in the form of small, dusky red, subconjunctival swellings or nodes, which usually appear in the ciliary region on the temporal side of the cornea, though patches may occur in any portion of the zone.

The conjunctival vessels over the patch are coarsely injected, and movable with the somewhat edematous conjunctiva. The episcleral vessels show a dusky congestion which is immovable. The elevation is sometimes tender to pressure and sometimes not, and there may or may not be much irritation and pain. In some cases of phlyctenular disease of the corneal margin it is difficult to decide between this affection and episcleritis; what appears to be a patch of the latter may develop into the former.

The disease runs a subacute course, reaching its height in about three weeks, then gradually disappears, and leaves a somewhat dull area of discoloration marking its former position. Relapses are frequent, both at the original seat or in new spots on the sclera, and these recurrences may happen again and again for months and even years. The cornea and uveal tract easily participate in the inflammation.

Cause.—Episcleritis and *episcleral nodes*, either solid or moderately soft in texture, are more frequent in women than in men in the author's experience. Patches of episcleritis of the character described occur in the eyes of those who are much exposed to the weather. In other cases superficial scleritis is caused by rheumatism, gout, tuberculosis, menstrual derangements, enterogenous auto-intoxication and focal infections in the teeth and tonsils, and also appears without discoverable cause. Well-marked episcleral (not conjunctival) congestion or episcleritis occurs in connection with disease of the accessory sinuses, especially the ethmoids.

In these forms of superficial scleritis the *prognosis* is good so far as sight is concerned, because deeper and adjacent structures are uninvolved, but unfavorable on account of the recurrences.

Treatment.—This consists in the use of atropin to allay pain and prevent any tendency to iritis, warm antiseptic collyria, and hot compresses. Dionin is of distinct service. In the chronic types eserin and pilocarpin, $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.0162–0.324 gm.) to the ounce (30 c.c.) of water, have a beneficial influence, provided no iritis is present. Subconjunctival injections of salt solution are useful, and similar injections of salicylate of sodium (2 per cent.) and of hetol (cinnamate of sodium) have been recommended (Pflüger). Massage with a salve of the yellow oxid of mercury is indicated in chronic cases, and it has been recommended to scarify the tumefaction, scrape it away with a sharp curet, or cauterize it repeatedly, in a superficial manner, with the actual cautery. Internally, salicylic acid and iodid of potassium are needed in rheumatic cases, and good results follow diaphoresis induced by pilocarpin or a Turkish bath. Menstrual and uterine disorders must be rectified, and the influence of intestinal sepsis eliminated as well as any areas of focal infection. If a tuberculous taint is discovered, injections of *tuberculin* are of service. Any error of refraction or anomaly of the exterior eye muscles should be corrected.

Fugacious Periodic Episcleritis.—This name has been given by Fuchs to a variety of relapsing episcleritis characterized by the appearance of one or more patches of episcleral injection or edema, of violaceous hue, lasting from two to eight days, and reappearing again at intervals of several weeks or even months, to go through the same course. The duration of the affection is usually about one year; it occurs most frequently in adults. Gout and rheumatism are associated constitutional conditions. The same affection was described years ago by Swan M. Burnett under the name of "Vasomotor Dilatation of the Vessels," and by Jonathan Hutchinson as "Hot Eye." The treatment is similar to that already recommended for episcleritis.

Scleritis may appear in the form of a *diffuse*, bluish-red injection, occupying the entire exposed portion of the sclera, very painful, unattended by secretion, save some increase in lacrimation, and liable to be mistaken for conjunctivitis or iritis; or in the form of *circumscribed* patches of violaceous tint, situated in the ciliary region, and somewhat resembling in appearance the forms of superficial or episcleral elevations just described, being, however, less sharply defined, so that the whole zone may be involved, but in unequal degree. Spicer calls attention to crescentic areas of infiltration in the cornea, separated from the patch of scleral inflammation by a band of clear cornea. In many cases of diffuse deep scleritis hard, whitish nodules develop in the inflamed tissue (*nodular scleritis*). Overgrowth of the infiltrated tissue may produce diffuse or circumscribed areas, which are called *hyperplastic scleritis*. The chief distinction between the *superficial* and *deep* forms of scleral inflammation is the almost invariable tendency of the latter to affect other portions of the eye—the cornea and uveal tract.

Pathology.—In episcleritis the infiltrating cells are found either in the superficial layers around the conjunctival vessels or in the deepest layers. The vessels are dilated, extravasations of blood are found, and often spots of necrosis and giant-cells. Usually the choroid and sclera are infiltrated and edematous.

The **causes** of deep scleritis are exposure to cold, rheumatism, gout, scrofula, vasomotor changes, and disturbances of the sexual apparatus, especially anomalies of menstruation. Young adults are most frequently attacked. The so-called *gummatous scleritis*, in which the patches are yellowish brown and translucent, is due to syphilis; and gonorrhea, if it is associated with synovitis, may cause the disorder. One form of scleritis may be the forerunner of parenchymatous keratitis. Deep scleritis is also seen in the subjects of congenital syphilis and tuberculosis. The trial of tuberculin in scleritis is often followed by a general as well as a local reaction. Excised nodules may show epithelioid cells and giant-cells, but no tubercle bacilli. *Tuberculous scleritis* is probably due to an infection derived from the aqueous arising from the filtration angle (Verhoeff). Tuberculosis of the sclera, according to Collomb, is secondary and should be regarded as a metastatic localization in the scleral vascular bundles surrounding the cornea. Finally, types of scleritis (sclerokeratitis) unassociated with any definite cause or diathesis are seen in young and middle-aged subjects, most commonly women, whose nutrition is depressed, and who may or may not have a tuberculous disposition or inheritance. Scleritis may be a metastatic inflammation, the original focus of infection being at some distant part of the body, for example, a rectal abscess, a felon, or a purulent sinusitis (Dupuy-Dutemps) and arise because of the more usual focal infections in the teeth, tonsils, and intestinal tract. Disturbances of the internal secretions are doubtless of etiologic influence in many cases.



Fig. 143.—Tuberculous sclerokeratitis, showing scleral nodules and characteristic triangular corneal infiltration (from a patient in the University Hospital).

Deep scleritis usually attacks both eyes, runs a chronic course, and may affect the iris (leading to closure of the pupil), ciliary body, choroid, vitreous (causing opacities), and the cornea. In prolonged cases of the disease dark scars remain after absorption of the products of the inflammation, which are unable to resist the intra-ocular pressure, and form elevations (*ectasia sclerœ*). Total scleral ectasia, or an enlargement of all the diameters of the globe, is seen in buphthalmos (page 442). Sometimes the whole anterior portion of the sclera becomes bluish or slaty

colored, is misshapen and elongated, and the cornea, which appears small, is poorly differentiated from it on account of the haziness of its margins.

Sclerokerato-iritis (*Scrofulous Scleritis; Anterior Choroiditis*).—This name is applied to the complicated scleritis referred to in the previous paragraph, and is characterized by chronicity, relapses, and involvement of the cornea and iris.

Beginning with a deep scleritis of the ciliary zone, the adjacent cornea becomes opaque and sometimes ulcerates; the iris is inflamed, posterior synechiæ form, and pain and congestion may be severe. After weeks the symptoms subside, the characteristic discolored area marks the former scleral disease, and haziness in the cornea indicates the seat of previous inflammation. Relapse takes place, with fresh scleritis, new corneal involvement, renewed iritis, or iridochoroiditis, and vitreous changes, and so on, until after many months, it may be, the disease comes to an end, leaving the sclera discolored and bulged, the cornea covered with patch-like opacities, the iris bound down by adhesions, the vitreous filled with opacities, and the eye practically deprived of vision.

Sclerotizing keratitis, referred to on page 306, is the name given to a patch of opacity in the deeper corneal layers, usually triangular in shape, with its base toward the patch of scleritis, which is its origin. After the subsidence of the scleritis a white or yellowish-white opacity remains directly in contact with the sclera. Instead of a single patch of this character, several small triangular areas may appear at the circumference of the cornea as the result of scleritis.

Treatment.—The treatment of scleritis and sclerokerato-iritis depends upon the type and stage of the disease and the presence or absence of definite cause. The elimination of the areas of focal infection (teeth, tonsils, etc.) constitutes an important part of the treatment. Locally, atropin, hot compresses, holocain, dionin, and boric acid lotion, and in painful cases leeches on the temple are suitable. Pilocarpin is valuable if iritis is not present. The eyes should be carefully protected by goggles. After the subsidence of acute symptoms massage of the eyeball may be tried. The use of the actual cautery has been mentioned. Subconjunctival saline injections are useful.

In rheumatic cases salol, the salicylates, the alkalis, and iodid of potassium are the most available remedies; in gout, carefully regulated diet, mineral waters—Buffalo, Poland, etc.—citrate of lithium, and colchicum, especially in the form of colchicin, are useful. Cod-liver oil, iodin preparations, iron, and sweats by means of pilocarpin, $\frac{1}{10}$ grain (0.00648 gm.) hypodermically, are also indicated. The diaphoretic measures are proper in any case, other things being equal, and, in place of pilocarpin, an electric cabinet or a Turkish bath may be the means of creating diaphoresis. In syphilis, bichlorid of mercury, inunctions of mercurial ointment, and arsphenamin are efficacious. Indeed, mercury is generally advantageous as a means of altering the nutrition of the part and preventing exudation into the uveal tract.

Disorders of menstruation should always be corrected. Finally, in the subjects of depressed nutrition, quinin, arsenic, and a general tonic regimen are required. Because many cases of scleritis are due to tuberculosis a tuberculin treatment (see page 352) is often indicated, and may be followed by most satisfactory results. Tests with tuberculin should always be made; a positive reaction is obtained in a number of instances. The internal administration of thyroid extract is indicated in some cases.

Annular Scleritis (*Brawny Infiltration of the Sclera*).—To this severe form of scleritis, which invariably affects the whole region around the cornea, J. Herbert Parsons, G. Derby, and Verhoeff have called renewed attention. Unlike ordinary scleritis, which usually attacks young adults, this affection is a disease of advanced age or, at least, of middle life. Both eyes are usually affected, though not to an equal extent. The disease is essentially chronic, and subject to periodic exacerbations and remissions. Verhoeff believes that syphilis is an important etiologic factor, although this cause is doubted by Schodtmann, who first described the disease. Gilbert has ascribed it to gout. The prognosis is most unfavorable, many of the eyes having been lost. The corneal margin is the essential site of the infiltration, from which region it spreads on both sides into the surrounding tissues, overlapping the cornea on the one side and extending as far as the equator of the eyeball posteriorly on the other. The swelling is usually gelatinous and succulent and has a brownish-red color. In addition to the involvement of the cornea, the uveal tract, especially the anterior part of the choroid and the ciliary body, are inflamed.

Posterior Scleritis.—In this affection, as described by Fuchs, the symptoms are edema of the lid, exophthalmos, conjunctival chemosis, and ophthalmoscopically the appearance of detachment of the retina, or of a gray cloudiness over the affected area. After the subsidence of the inflammation changes in the retina remain. Secondary iridocyclitis may arise. According to Coats, the disease depends upon blocking of one of the larger ciliary arteries and consequent infarction of the inner layers of the sclera, choroid, and retina.

Staphyloma of the sclera (*ectasia of the sclera*) has been divided by systematic writers into *partial* and *total* ectasia and *anterior*, *equatorial*, and *posterior* staphyloma, according to the situation of the lesion. Posterior staphyloma is detected with the ophthalmoscope in a highly myopic eye (see page 143) and consists of a thinning and bulging of the sclera usually at the outer side of the optic nerve entrance (*posterior staphyloma of Scarpa*). In association with coloboma of the choroid (page 383) below the posterior pole of the eye there may be a scleral ectasia (*posterior staphyloma of Ammon*).

It is evident that all bulging of the sclera depends upon a disturbance between the resistance of the sclera and the intra-ocular tension, but it is not evident in all cases whether the process which originated the trouble began in the underlying tissue or in the scleral structure itself. One or more darkly tinted swellings may arise in the ciliary region

and sometimes entirely surround the eyeball (*ciliary staphyloma*) or in the region in front of the ciliary body, that is, between it and the edge of the cornea (*intercalary staphyloma*), one sometimes occurring in advance of each rectus tendon; or, finally, the staphylomatous swellings may exist at the equator in the region of the vena vorticosa and are not noticeable unless the eye is rotated strongly in one direction or another. A general enlargement of the scleral coat is seen, for example, in hydrophthalmos (page 442) or where in young subjects a combination of staphyloma of the cornea (page 287) and anterior scleral staphyloma (*total ectasia*) exists.

The following **causes** may originate scleral staphyloma: Chronic glaucoma, old kerato-iritis and closure of the pupil, recurring scleritis and sclerokerato-iritis, inflammation of the ciliary body, thinning of the scleral coat by repeated attacks of inflammation, tumors, and wounds closed by non-resisting scars.

Treatment.—A single scleral staphyloma may not destroy vision. If the intra-ocular tension is increased, an iridectomy is indicated. If the eye is useless, enucleation or one of its substitutes may be necessary.

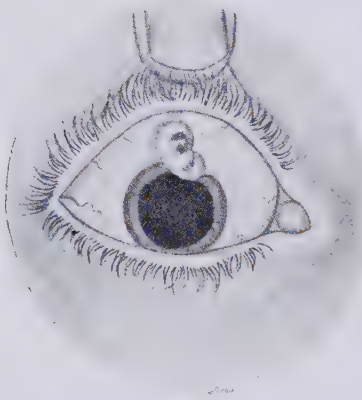


FIG. 144.—Cyst of the corneoscleral junction (Philadelphia General Hospital).

Abscess and **ulcers of the sclera** are exceedingly uncommon. Abscess in the scleral tissue may result from an infected wound and has been seen in connection with certain specific and contagious diseases—*e. g.*, glanders. Metastatic scleral infection has been recorded (see page 323).

Ulcer of the episcleral tissue has been described in association with tuberculosis. A tumor, gumma, or tubercle of another region of the eye may break down and ulcerate into the sclera.

Tumors of the sclera are rare growths. The following have been described: Fibroma, fibrochondroma, enchondroma, and osteoma.

Primary sarcoma, if it exists, must be rare; recently a case has been recorded; secondary sarcoma, carcinoma, and glioma have been reported. Gumma of the sclera has been described, and tubercle may invade it from the uveal tract. A few *scleral cysts* have been recorded. A recent study of epithelial cysts of the sclera, by Charles Goulden and M. H. Whiting, indicates that they may be congenital or may be due to injury. A certain number of them are encountered for which an etiologic factor cannot be determined. Small primary scleral growths may be dissected from their beds, and the wounds closed by sutures which pass through the conjunctiva (Fig. 144).

Injuries of the Sclera.—Wounds of the sclera may be caused by

a sharp implement (knife, scissors, broken glass, etc.) or foreign body (chip of iron or steel, fragment of shrapnel, bullet, etc.), or they may result from a blow on the bulbus on the inner side and above, more rarely downward and out (impact of a flying object, *e. g.*, golf ball, thrust of a blunt object, *e. g.*, a cow's horn, violent contact with a stationary object, *e. g.*, edge of a door), causing *rupture of the sclera* which is usually found 3 mm. from, and concentric with, the corneal margin (E. T. Collins). The rupture may be exposed through a rent in the conjunctiva, and is then said to be "compound," or it may be concealed by the conjunctiva, which is untorn. It may be *direct*, that is, rupture takes place at the point of impact, or *indirect*, that is, the rupture takes place at some point other than at the point of impact. Indirect ruptures of the sclera occur in the vicinity of the cornea and are more or less concentric with it, because here, due to the presence of Schlemm's canal and the penetrating anterior ciliary veins, there is an area of poor resistance. A blow may also *rupture the cornea*. Corneal tears, according to L. Müller, are more common in young people than scleral ruptures. Small ruptures at the limbus or within the corneal margin are usually associated with iris prolapse. *Incomplete ruptures* of the sclera have been observed (Fuchs) made manifest by a bluish hue near the limbus, which later becomes ectatic.

A wound of the sclera often extends into the cornea, cutting through the ciliary body, that is, through the corneoscleral junction; *prolapse of the iris* or of it and some of the tissue of the ciliary body is almost always a complication.

If the wound has *perforated* the sclera, two complications are liable to be present: loss of a portion of the contents of the globe and injury of the inner coats, and the introduction into the eye of septic material which will cause destructive inflammation.

Symptoms.—A perforating wound of the sclera, if sufficiently large, causes loss in the tension of the globe, hemorrhage into the vitreous, or, it may be, into the anterior chamber, and the appearance of dark tissue in the wound, representing, according to its situation, portions of the choroid or ciliary body; a bead of vitreous is likely to be present. A small perforating scleral wound may be hidden by the overlying contused and swollen conjunctiva. Usually the intra-ocular lesion is lower than normal. It must be remembered that although *hypotony* is a symptom of penetrating wound or rupture of the sclera, it may also occur as the result of contusion of the globe without rupture; hypotony does not prove that rupture is present. *Rupture of the sclera* is commonly associated with grave lesions in other portions of the eye —separation of the retina, tears in the choroid and iris, and dislocation or expulsion of the lens. To the extensive disorganization of the eyeball, and large wounds or ruptures of the sclera so frequently observed during the past war the term "shattered eye" was often applied.

Prognosis.—This depends upon (1) the extent and situation of the wound and amount of escape of vitreous; (2) the presence or absence of septic material upon the implement or body which inflicted the

injury; (3) whether a foreign body has remained within the globe; and (4) the character of the foreign body which may have entered. It is evident that even a trifling perforating wound, unattended by loss of vitreous or prolapse of the inner coats, may be a point of entrance of infection.

Treatment.—Having carefully ascertained that no foreign body is within the globe, the eye should be disinfected with a solution of bichlorid of mercury (1 : 5000), and the edges of the wound, after all foreign substances have been removed, penciled with a stronger solution of the same drug (1 : 2000) or with a 5 per cent. solution of iodin. The overlying conjunctiva is next drawn together with several fine sutures. The eye is closed with an antiseptic compressing bandage and the patient is put to bed. Iced compresses are an advantage during the early stages of the treatment. At the end of forty-eight hours the wound may be inspected and the dressings renewed. In larger wounds the sutures (sterile silk or catgut) are passed directly through the sclera, care being taken to avoid the choroid; the procedure, although not universally employed, in many cases possesses distinct advantages; the sutures may be removed at the end of a week if the healing has progressed favorably. Some surgeons advise the introduction of iodoform before the application of the bandage. In some instances, in spite of kind healing of the scleral wound, there are subsequent detachment of the retina, vitreous change, and shrinking of the eyeball, but occasionally apparently hopelessly injured eyes may be saved by careful conservative aseptic surgery. As *tetanus*, especially after earth contamination, may follow penetrating wounds of the eye antitetanic serum should be administered.

In the event of a scleral wound being extensive, with much loss of vitreous and collapse of the coats, especially if the ciliary body is involved and sight practically gone, or if the endeavors to remove the foreign body have been unsuccessful, enucleation should be performed to avoid the dangers of sympathetic inflammation in the fellow eye.

Foreign Bodies.[1]—If the wounding substance has been small—*e. g.*, a chip of steel, a splinter of glass, a particle of dynamite cap, fragment of shrapnel or a bullet—endeavor should be made to ascertain whether this has penetrated the globe and remained within it, or has passed entirely through the eyeball and buried itself in the tissues of the orbit. Foreign bodies may be embedded in any of the structures of the eye and are frequently found in the vitreous. If loose, they tend to gravitate to the lowest part of the vitreous and rest upon the posterior part of the ciliary body (E. T. Collins).

Double perforation of the eye is not uncommon as the result of a bullet wound or one caused by the explosion of a dynamite cap, but less frequent if the foreign body is a chip of iron or steel. In rare instances the perforation of the posterior scleral wall has been discovered

[1] It is convenient to discuss in this place the treatment of *foreign bodies* which are lodged in the *vitreous* or any of the internal ocular coats after having penetrated the sclera covering.

with the aid of the ophthalmoscope, but since the introduction of *x*-ray examination the diagnosis is rendered comparatively easy, and the radiographs should show whether the foreign body has passed entirely through the posterior scleral wall or is embedded partly within and partly without the scleral covering.

According to Leber, perforating injuries of the eye caused by pieces of *copper* may result in purulent inflammation merely by the chemic action of the metal; if infection is absent, an attempt to remove the body may be made, and, if successful, the eye saved, even if inflammation has begun.

Foreign bodies may be tolerated for long periods of time, with good vision, in the background of the eye, but can never be trusted, especially if located in the uveal tract; they are liable to cause degenerative changes. However, small *fragments of glass* have remained for years within the globe without creating untoward symptoms and the lens is more tolerant of foreign bodies than the other internal ocular tissues. Sometimes foreign bodies are embedded in the external scleral walls; if anteriorly placed they are readily removed with a magnet if they are either iron or steel.

Unfortunately, blood in the vitreous and anterior chamber, or opacity of the lens, is apt to obscure the media to such a degree that ophthalmoscopic examination is not of much service; but if the media are clear, this method may be the means of detecting the foreign body. *Air bubbles in the vitreous* are suggestive, but not pathognomonic of a foreign body in the globe. An attempt at locating the body may be made by observing the situation of the wound, the condition of the capsule of the lens, the probable direction which the foreign substance took on making its entrance, by a search for points of tenderness and for a scotoma in the field of vision. If there is any doubt, a skiagraphic examination should be undertaken, and in the majority of instances (pieces of wood excepted) the Roentgen rays readily reveal the presence and position of the foreign body. Of the various methods devised for this purpose, the one elaborated by W. M. Sweet, in the opinion of the author, is most satisfactory. The method of McKenzie Davidson and Dixon's modification of the Sweet method are also excellent.

Having satisfied himself of the presence and position of a *non-metallic foreign body within the globe*, the surgeon may attempt to extract it through the original wound with delicate, carefully disinfected forceps, or through a new wound made in the most favorable situation, guided if possible by simultaneous ophthalmoscopic examination. But in some instances a small fragment of sterile glass, for example, difficult or almost impossible to reach should be allowed to remain rather than to make matters worse by the frequent introduction of instruments into the vitreous chamber in the effort to extract it. During the past war a few non-metallic foreign bodies were located by a specially devised fluoroscope and extracted through a scleral incision, but such fortunate operative results were rare.

If the foreign body is composed of iron or steel and its presence

cannot be detected on account of opacities in the media, a diagnosis may be made, as was first suggested by T. R. Pooley, with the magnetic needle. Useful instruments have been constructed on this principle by Asmus and Hirschberg, and are known as *sideroscopes*, with which a properly protected magnetic needle is brought near different portions of the eye in succession, and any deviation of the needle carefully noted. Where the deviation is greatest there is reason to suspect the foreign body exists.

A large magnet may also be employed for diagnostic purposes, the dislodgment of the foreign particle giving rise to a localized spot of pain or point of bulging, but great care must be exercised in attempting this procedure, as sudden movement of the foreign body may



Fig. 145.—Foreign body in the vitreous.

cause serious intra-ocular lesions, hemorrhage, tearing of the iris, etc. Moreover, as has been pointed out by Hirschberg, the absence of pain when the eye is approached by the magnet does not surely exclude the presence of a foreign body.

The most satisfactory method in a doubtful case, and where the media are so obscured that ophthalmoscopic examination is impossible, is the employment of the *x*-rays, which, if properly used according to Sweet's method (see Appendix, page 803), have, in the author's experience, never failed to give exact information of the position of the foreign substance. When this has been determined, the body should be extracted by means of an electromagnet. In former times it was much the practice to introduce the extension point of a magnet, for example,

the Hirschberg model, either through the original entrance-wound or through one made for that purpose, as far into the vitreous as was necessary to attract the splinter of iron or steel from its position. This method has been abandoned for others which are much more satisfactory in that the introduction of an instrument into the vitreous is avoided. The magnets most in use are *giant magnets* (Haab's model and its modifications—an instrument unsurpassed in excellence; Volkmann's model; Hirschberg's model); the *Innenpol magnet* designed by Mellinger, the patient's head being placed within a large electric coil, while the passage of the current magnetizes both the foreign body in the eye and the iron instrument held in front of the eye to attract it; and *large*, or *sling-magnets*, of which those designed by Sweet, F. Parker, Lancaster, and Lister are types.

Whether intra-ocular metallic foreign bodies shall be removed along the anterior or posterior route must be settled by the surgeon in charge of the case. The author is in favor of the posterior route after *x*-ray localization of the fragment, save only where there is a large, fresh wound of entrance and a body of considerable size, and in the case of very small bodies in the forward regions of the eye. Points of importance are that the operation shall be done as soon as possible after the accident, that not infrequently small foreign bodies penetrate and leave no external evidence of the point of entrance—hence the importance of *x*-ray examination of any eye which has been exposed to the possibility of such an accident. The wound of entrance, or one made during the operation of extraction of the foreign body, must be carefully covered with a conjunctival flap (see Conjunctivoplasty).

There are three routes along which the foreign body may be attracted by the magnet: (*a*) Through the wound of entrance, advisable if the body is of large size; (*b*) from the regions posterior to the plane of the iris, *i. e.*, the ciliary body, lens, vitreous chamber (the usual situation), choroid or retina, into the anterior chamber, the so-called *anterior route;* (*c*) through a small opening made in the sclera, its position being determined as the one nearest to the situation of the foreign body by means of *x*-ray localization, the so-called *scleral* or *posterior route.*

During the past war *x*-ray examination often was not possible or the delay entailed was not deemed advisable. Haab strongly condemns the extraction of steel or iron from the interior of the eye through a scleral incision, and his opinion in this respect is shared by many surgeons, and Parsons has advanced reasons from the pathologic standpoint in opposition to a scleral route. Charles Goulden examined the records of 118 extractions of metallic foreign bodies from the eye, and found that the worst ultimate results occurred if the body entered through the sclera or if it was removed by scleral puncture. The author's preference has been stated. For methods of operating and a further duscussion of this subject see pages 750, 752.

If infection has already begun when the patient is seen, and the condition of the eye is not so hopeless that immediate enucleation

is necessary, various methods have been tried to check the purulent process. If, for example, the wound of entrance has been through the cornea, and the anterior chamber contains pus, this may be evacuated, and, as Haab recommends, small rods of sterilized iodoform, one or two, according to circumstances, may be introduced within this chamber. He also, in like manner, introduces these rods directly into the vitreous if infection has begun in that region, and reports successes. This method of intra-ocular disinfection has been sharply criticized—Krause, for example, believing that iodoform, either in the form of powder or rods, is unable to influence favorably beginning infection, but that, on the contrary, of itself it may produce pathologic changes, and represents a method inferior to other well-known procedures for the relief of infected wounds—for example, that of Schirmer, who recommended mercury by inunctions. The author has tried the iodoform method in several cases with indifferent success, and prefers the use of mercury, especially calomel, in repeated, properly guarded, doses. Drainage of the anterior chamber, expression of the lens, and thorough irrigation of the posterior chamber with salt solution have seemed to be of service in a few cases in the author's practice. Constant iced compresses are of value. Van Millingen has suggested the trial of endocular cauterization in these circumstances—that is, the introduction into an infected scleral wound of a galvanocautery point, if necessary, even into the vitreous, and the cauterization of all surrounding tissue.

Prognosis.—This is always grave, but by means of the methods just detailed many eyes have been saved, and some with useful vision. The important point is to operate as soon as possible after the accident —*i. e.*, before the foreign substance has become incarcerated in the tissues and covered by lymph. The prognosis is more favorable if the foreign bodies are situated in the vitreous than if they are entangled in the ciliary body or choroid. Goulden's results have been referred to (see page 331). If judicious efforts have failed to extract a foreign body from the interior of the eye, or if infection has proceeded beyond the reasonable hope of recovery, enucleation or evisceration usually is necessary.

If a particle of iron remains for some time in the eye, there is a deposit of iron pigment in its tissues which gives rise to a condition known as *siderosis bulbi*, characterized by a peculiar greenish-yellow or yellowish-brown discoloration of the iris and cornea, and a circle of brown dots beneath the capsule of the lens. The pigmentation may be due to the iron derived from the foreign body (xenogenous pigmentation) or to hemosiderin derived from blood (hematogenous pigmentation). Sometimes the iris regains its original color after removal of the foreign body and, occasionally, even if it remains within the eye.

Congenital pigmentation of the sclera (*melanosis sclerœ*) occurs both in spots and as a more diffuse discoloration. The spots are more common in the upper portion, and may be associated with pigment changes in the iris and choroid. Pigment spots on the sclera have been observed in certain diseases—*e. g.*, Addison's disease—and some-

times are exactly symmetric, situated near the margin of the cornea (see also page 320).

Blue scleras may be associated with inherited syphilis and exhibit a remarkable hereditary transmission. In addition to the leaden color of the sclera, there may be conical cornea and congenital opacity of the cornea, that is, *embryotoxon*. Harman traced in five generations of one family 55 members, of whom 31 showed this congenital peculiarity. Transmission occurs through affected mothers. Persons with blue scleras are peculiarly liable to bony fractures.

CHAPTER IX

DISEASES OF THE IRIS AND CILIARY BODY

Congenital Anomalies.—*Heterochromia*, or the condition in which the color of one iris is different from that of the other, is a peculiarity which may be without pathologic significance, but in many instances the signs of cyclitis in the lighter colored eye are evident, and this eye is liable to cataract formation (see page 55).

Corectopia, a term applied to an eccentric position of the pupil, must not be confounded with cases of true coloboma of the iris, presently to be described. The grade of corectopia may vary from a slight increase of the normal eccentric position of the pupil below and to the inner side, to those cases in which the whole pupil is displaced toward the border of the cornea. The latter variety is a very unusual phenomenon. This complete shifting of the normal position of the pupil has been ascribed either to an essential malformation or to the result of a *fetal iritis*. Both eyes may be affected symmetrically, and several members of the same family may present the defect.



Fig. 146.—Polycoria.

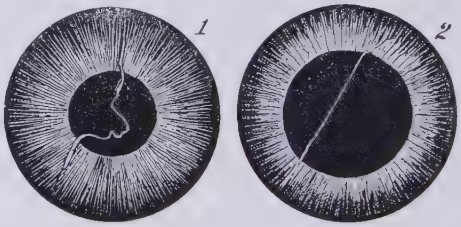


Fig. 147.—Persistent pupillary membrane: 1, Pupil contracted; 2, pupil dilated (Wickerkiewicz).

Polycoria, or a multiplicity of pupils, is a rare anomaly. The abnormal pupils may be situated in the immediate neighborhood of the normal pupil, separated from one another by a narrow band of iris tissue, or the increased number of pupils may be the result of crossing strands of persisting pupillary membrane (Fig. 146, see also page 340). An opening which exists at the ciliary margin of the iris has been described, and is probably due to a *congenital iridodialysis*.

Persistent pupillary membrane results from an incomplete elimination of the membrane which covers the anterior surface of the lens during fetal life, and which usually disappears in the seventh month, although it may remain as late as the end of intra-uterine life, and even in the first month after birth.

Accurately speaking, the pupillary membrane is a specialized portion of the *capsulopupillary* covering. The name of pupillary membrane alone is applicable to those cases in which threads attached to the

small circle of the iris pass diametrically or cord-wise across the pupil, to be inserted elsewhere in the *corona* (Fig. 147). Usually the fibers proceed from the anterior surface of the iris across the pupil, either singly or in groups of three or more strands. Sometimes the fibers remain separated; sometimes they grow together in front of the anterior capsule or unite in the form of a variously colored plaque, adherent to the capsule of the lens (*capsulopupillary membrane*). Persistent pupillary membrane is more common in one than in both eyes; of 68 cases observed by Stephenson, 13 were bilateral and 55 unilateral.

Capsulopupillary tags are not infrequently mistaken for the synechiæ due to iritis; indeed, the association of the two has been observed. No difficulty, however, should arise, because the action of the pupil is not impeded by the presence of these vestigial anomalies and the response to a mydriatic is normal. The appearance is not often detected until some other disorder calls for an ophthalmoscopic examination, because vision is not seriously or at all impaired. Oblique illumination or examination with a loupe or corneal microscope will readily demon-

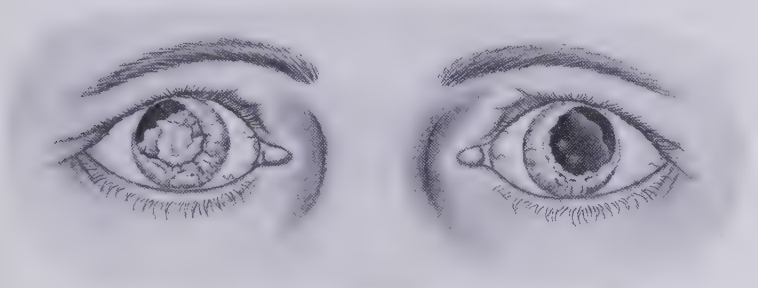


FIG. 148.—Bilateral coloboma of iris, upward and outward, and cataract (University Hospital).

strate the remains of pupillary membrane. Naturally, slit-lamp investigation is especially valuable in this respect.

Coloboma of the iris is a fissure of this membrane which in a general way resembles an artificial pupil made by iridectomy. The anomaly is more frequent in both eyes than in a single eye. Where the defect is unilateral, the anomaly is usually found on the left side. The situation of the fissure is generally downward or downward and inward. Exceptions to this rule have been observed; indeed, numerous atypical forms have been observed, the defect being placed outward, inward, upward, down-and-out, up-and-in, and up-and-out.

The coloboma may extend across the whole iris (*complete coloboma*), or stop at a certain distance from the ciliary margin (*incomplete coloboma*). In addition, the so-called *pseudocoloboma* is described, which may be looked upon as a form of heterochromia of the iris, or indicates the last remains of the ocular fissure which is tending toward closure, and which appears as a small stripe, somewhat granular, and differentiated from the rest of the iris by its brighter color. In "bridge colo-

boma" the borders of the cleft are united by a narrow pigmented or colorless band of fibers.

Coloboma of the iris, often hereditary, is frequently associated with similar defects in the choroid, and also with microphthalmos, congenital cataract, fissure of the eyelids, lips, and palate. Upward coloboma of both irides, and of the choroid, a rare if not unique anomaly, has been observed (Sanford Gifford). Coloboma of the iris has been attributed to an arrest of development, the result of incomplete closure of the *choroidal fissure;* but Lang and Treacher Collins believe that the defect is caused by a partial abnormal adhesion or late separation of the lens and cornea, the iris failing to develop in that portion of the area which is involved. If the abnormal adhesion or late separation is complete, irideremia results.

Irideremia, or congenital absence of the iris, occurs both in a *partial* and a *complete* form.

Total congenital irideremia is almost invariably bilateral. It is frequently associated with other anomalies of the globe—partial or complete cataract, dislocation of the lens, nystagmus, strabismus, departures from the normal curvature of the cornea, or annular opacities in its periphery and atrophy of the optic nerve. In a majority of instances there is a marked hereditary tendency.

Congenital ectropion of the uvea consists in a round mass of dark color projecting from the margin of the pupil, bending around to the anterior border of the iris. A similar formation is proper to the eye of the horse and is frequently seen in the cow. This appearance has sometimes been described as a *papilloma of the iris;* it is not, however, a neoplasm, but a congenital ectropion of the uvea.

Cysts, nevi, and *atrophies of the iris* occur as congenital defects, and *congenital aplasia of the anterior layers of the iris* has been observed.

IRITIS

Under the general term *iritis* are included various types of inflammation of the iris, but the intimate anatomic connection of the iris and ciliary body frequently unites them in the inflammatory affections to which they are subject. In other words, iritis rarely if ever exists without cyclitis and *vice versa*—hence the term *iridocyclitis.* The descriptive word *uveitis* is generally employed to indicate an involvement of the entire uveal tract in the inflammatory process (page 361).

Naturally, the process may predominate in the iris (iritis), or in the ciliary body (cyclitis), hence it is customary and not inadvisable to indicate the symptoms in these circumstances in separate descriptions (page 359).

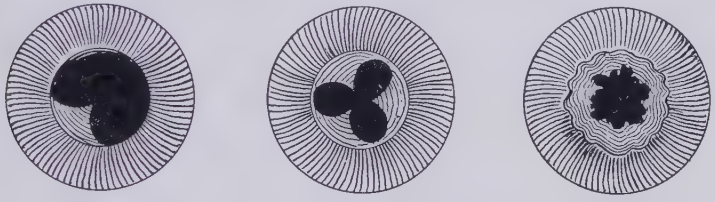
Causes.—Iritis or iridocyclitis may depend upon constitutional disorders, infections, toxins, and traumatism, or upon disease in other portions of the eye. Hence, it is usually stated that iritis is either *primary* or *secondary.* To those cases of iritis which apparently originate independently of injury, or of an ocular or constitutional disorder, the name *idiopathic* was formerly applied, a term which should be

eliminated, although, unfortunately, we are unable always to decide what exactly is the causative factor in each case. Iritis is also divided, according to its supposed etiology, into *syphilitic, rheumatic, gouty, gonorrheal, diabetic, tuberculous, scrofulous, septic, autotoxemic* or *toxemic, cachectic, traumatic,* and *sympathetic* iritis.

Symptoms.—1. *Hyperemia of the iris* usually, but not invariably, is the first sign of iritis.

It is recognized by *change in color,* a blue iris becoming greenish; a brown iris, a reddish brown; by contraction of the pupil, which responds sluggishly or not at all, to the changes of shade and light, and is slowly affected by a mydriatic, the effects of which are much less permanent than in the healthy iris; and by slight pericorneal injection. It is associated with certain acute affections of the eye, for example, conjunctivitis, trachoma, keratitis and scleritis, as a precursor of inflammation.

2. *Pericorneal injection,* due to congestion of the non-perforating branches of the ciliary vessels (System II), producing a fine pink zone surrounding the cornea known as "ciliary congestion," or the "circumcorneal zone." In severe cases there may be distention of the pos-



A *B* *C*

FIG. 149.—Various forms of posterior synechiæ: *A*, Single attachment; *B*, multiple attachment forming the so-called "ace-of-clubs" pupil; *C*, irregular annular attachments. (Sichel.)

terior conjunctival vessels, and slight chemosis of the conjunctiva (see also page 50).

3. *Miosis,* or contraction of the pupil, due partly to hyperemia as already noted and spasm of the sphincter, and partly to irritation of the peripheral nerve filaments. The reaction of the pupil to the influence of light and mydriatics is diminished or lost. In a certain number of cases, according to Herbert, there is slight dilatation of the pupil as compared with the unaffected eye. This primary dilatation of the pupil, with preservation of its reaction, is a noteworthy symptom in so-called rheumatic iritis (Krückmann; see also page 346).

4. The *formation of posterior synechiæ,* or attachments between the layer of pigment covering the posterior surface of the iris and the capsule of the lens. They are demonstrable by the instillation of a mydriatic, which produces an irregular dilatation of the pupil, certain portions of the pupillary margin of the iris being held back by somewhat tongue-shaped projections attached to the lens-capsule, and may be readily studied by means of oblique illumination and a loupe or corneal microscope, and especially with the aid of the Gullstrand

diaphragm lamp. The tags protruding into the pupil space usually have a brownish, sometimes a grayish, color.

The attachments may vary in size, firmness, and number; being either narrow and thread-like, broad and dense, single or multiple, or even extending all around and pinning down the margin of the iris in an annular manner (*annular posterior synechiæ*). In association with the synechiæ there may be an exudation of false membrane covering the whole pupillary space (*pupillary membrane* or *exudation*).

5. *Irregularities in the surface of the iris*, due to local swellings, accumulations of exudation, deposits of fibrin, or the formation of nodules.

6. *Haziness of the cornea* or deposits upon its posterior surface. According to H. Friedenwald, the cornea is affected in every case of iritis, that is, there are deposits on Descemet's membrane or infiltrations in the substantia propria. The former are constant, the latter occasional; E. C. Ellett describes a grill-like figure of straight translucent lines, usually at right angles to each other, as an early symptom of iritis.

7. *Changes in the character of the aqueous humor*—(1) Slight or considerable turbidity; (2) pus; (3) blood; and (4) occasionally exudation; slit-lamp microscopy reveals in certain cases strands and flakes of exudation within the cloudy anterior chamber (Vogt).

In addition to the symptoms just detailed there are *subjective* signs more or less peculiar to iritis.

1. *Pain.*—This is situated first in the eyeball, and is known as "ciliary pain," and second, in the brow and temple, sometimes quite sharply defined in the distribution of the supra-orbital nerve, very severe, throbbing, and stabbing in character, and with marked increase in severity during the night. Occasionally the nasal and infra-orbital regions are the painful areas. Pain in the teeth is not uncommon.

2. *Disturbance of Vision.*—This is in direct proportion to the amount of cloudiness which has occurred in the media. Decided impairment of visual acuteness denotes extension of the disease to the ciliary body or deeper structures.

During iritis, *transient myopia* and *astigmatism* are commonly present. Especially in the plastic types of the disease, even after full pupillary dilatation, an increase in the refractive power is demonstrable. Although there are changes in the corneal curvature the bulk of the ametropic change in such cases is due to perversion of the lens action from *spastic accommodation* (Koller) as the result of ciliary irritation.

3. *Tenderness of the Globe.*—This is often present in uncomplicated iritis, but if severe, it suggests inflammation of the ciliary body.

4. *Photophobia and Lacrimation.*—These symptoms vary considerably in degree, being almost or quite absent in some varieties, and severe in those of acute and violent onset.

5. *Malaise*, fever, nausea, and marked depression occasionally are experienced by the patient, the last often being the result of prolonged pain and insomnia.

Diagnosis.—The salient symptoms of iritis just detailed are sufficient for the purpose of diagnosis; nevertheless, it is not uncommon to

find a case of iritis mistaken for some other external inflammation. In ordinary conjunctivitis the absence of pupillary changes (synechiæ), the character of the conjunctival congestion (page 50) and that of the secretion (mucous or purulent) distinguish it from iritis. In the earliest stages of iritis, before definite signs are in evidence and there are only hyperemia and iris cramp, the diagnosis is at times difficult. Homatropin solution (2 per cent.) may be used to test the reaction of the iris to its influence; or euphthalmin and cocain may be similarly employed, which combination (10 and 4 per cent. respectively), even if glaucoma threatens, would be permissible as the effect could readily be neutralized by instilling a 1 per cent. solution of pilocarpin. It must not be forgotten that iritis and conjunctivitis may be coincident and that the latter condition, or at least a conjunctival hyperemia, may antedate an inflammation of the iris (page 346). Slit-lamp investigation reveals very early changes in the iris; for example, slight lesions, distending blood-vessels, and local swellings.

A diffuse scleritis somewhat resembles in its color the zone of pericorneal injection more or less characteristic of iritis, which, indeed, may be a complicating symptom of the disease. Acute glaucoma may be mistaken for acute iritis (for the distinguishing points see page 433).

Course, Complications, and Prognosis.—An iritis may pursue an acute course, reaching its termination in four to eight weeks, or be sluggish from its onset and last, in a slow and an insidious inflammation, for long periods of time. The termination of an iritis may be entirely favorable. The inflammatory adhesions disappear, and the iris regains complete mobility, only a few traces of iris pigment being seen on the capsule of the lens. On the other hand, more or less complete attachment causing distortion and inequality of the pupil (consult Fig. 149) may remain; or deposits of exudation may directly occlude the pupil and lie upon the capsule of the lens; or the tissue of the iris may show areas of atrophy and exhibit a bleached or grayish aspect.

The binding down of the iris throughout the whole extent of its pupillary edge, although the pupil itself remains clear, is denominated *exclusion* or *seclusion of the pupil;* if the pupil space is filled with the products of exudation, lying upon the capsule of the lens, the term *occlusion of the pupil* is applied. Extensive or annular synechiæ may be responsible for obliteration of the angle of the anterior chamber; the iris, owing to the exudation behind it, is bulged forward except around its pupillary margin, which is bound down, so that a crater-like depression is evident, and the appearance denominated *iris bombé* is present. This condition may cause increased tension, secondary glaucoma, and even shrinking of the vitreous, detachment of the retina, and atrophy of the eyeball unless the communication between the anterior and posterior chambers of the eye is restored by operative measures (Figs. 150 and 151). Repeated attacks of iritis, as well as chronic inflammation of the iris, may cause *atrophy of the tissue.* In these circumstances the color of the iris is gray, the markings of its surface disappear,

dilated blood-vessels and reddish patches appear, small holes develop in its tissues, the pupil margin is thin, and its tissue is friable (Fig. 152).

Other causes of atrophy of the iris (conveniently referred to in this paragraph) are increased intra-ocular tension (glaucoma), traction due to iris-inclusion in scar tissue, and pressure, for example by a fragment of lens material undergoing absorption. A form of progressive



FIG. 150.—Exclusion and occlusion of pupil with exudation behind iris following gummatous iritis; compare with Fig. 151 (from a patient in the Philadelphia General Hospital).

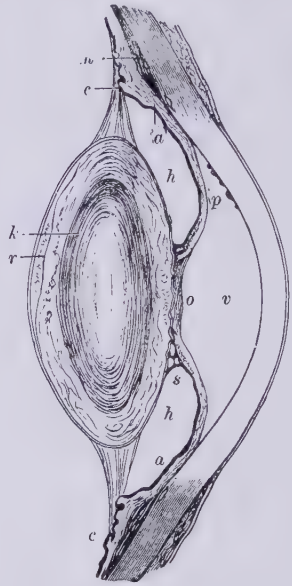


FIG. 151.—Exclusion and occlusion of the pupil. The iris is adherent by its entire pupillary margin to the lens. The posterior chamber (*h*) is thus made deeper, the anterior chamber (*v*) shallower, especially where the root of the iris (*a*) is pressed against the cornea. The retinal pigment is beginning to separate at *s*. The pupil is closed by an exudate membrane, *o*. In the lower part of the anterior chamber there is matter (*p*) precipitated upon the posterior surface of the cornea. The cortex of the lens has become cataractous (*r*); the nucleus (*k*) is unaltered. (Fuchs.)

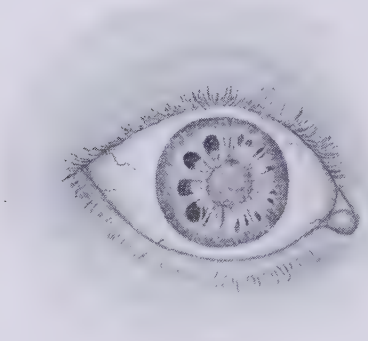


FIG. 152.—Atrophy of iris following inflammation (from patient in the University Hospital).

essential atrophy of all layers of the iris has been described (C. A. Wood, Harms, Zentmayer, Larsson, and the author). Periods of increased intra-ocular tension are evident and the eyes become glaucomatous. Wood did not consider that glaucoma was an essential part of the affection, although it was the end process in his patient. The anatomic examination revealed, in addition to glaucoma and iris atrophy, a fibrinoplastic cyclitis.

The following tissues of the eyes may become involved during the course of an iritis: The cornea (*keratitis punctata*); the ciliary body (*iridocyclitis*); the crystalline lens (*cataract*, especially *cataracta accreta*, in which the iris and lens are fastened together); the choroid (*iridochoroiditis*); the vitreous (*exudation into the vitreous, hyalitis*); and the optic nerve and retina (*hyperemia, retinitis, optic neuritis*). With these facts in mind, and with the tendency of certain types of the disease to relapse, a prognosis must be guarded, but in uncomplicated iritis, seen early and properly treated, a perfect result may be obtained in the large majority of cases. *Relapses of iritis* are often attributed to pos-

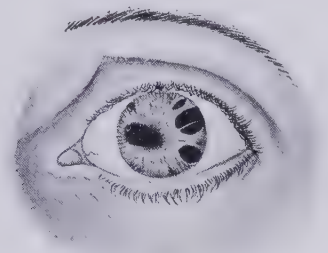


FIG. 153.

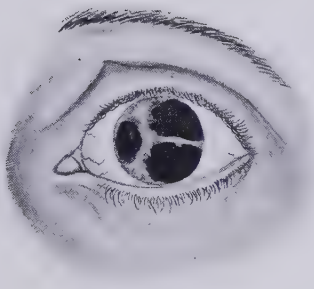


FIG. 154.

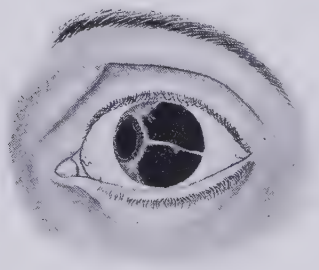


FIG. 155.

FIGS. 153–155.—Essential progressive atrophy of the iris, showing gradual elaboration of the process. The eye developed glaucoma.

terior synechiæ, but, as Fuchs points out, are not due to them, but to the continuance of the cause of the affection.

Pathology.—Systematic writers at one time were accustomed to divide iritis into three varieties: *plastic*, *parenchymatous*, and *serous* iritis. A more accurate classification from the pathologic standpoint is *acute*, *chronic*, *purulent*, and *nodular* iritis. In general terms, in iritis it may be said that the iris is thickened and infiltrated with round cells, which are collected either along the line of the vessels or in circumscribed nodules. The vessel walls are thickened and small hemorrhages occur in the tissue. The exudation in the anterior chamber consists of round cells, mixed with fibrin and pigment granules. Pigment of this

character may be deposited on the posterior surface of the cornea. In many cases the inflammatory products are completely absorbed, but should they become abundant, they organize, forming a layer of connective tissue which covers the iris and binds it to the lens, occluding the pupil in the manner already described. The exudation in the iris likewise organizes, and the atrophic iris shows obliterated and thickened vessels, clumping of pigment granules, and an entire absence of iris-stroma. *Purulent* iritis due to infection with micro-organisms is followed by panophthalmitis. This infection may come from without, for example, in perforating injuries, and is called *ectogenous infection;* or from within, as, for instance, in metastatic processes, and is called *endogenous infection.* In *nodular* iritis the nodes are formed of aggregations of lymphocytes. It is often difficult to distinguish between the varieties of iritis from the histologic standpoint, as they merge one into the other. If the exudation is poor in cells and fibrin, and the iris tissue shows cellular infiltration, there is serous iritis (not to be confounded with the cyclitis clinically called *serous iritis*); if the exudation is rich in fibrin but poor in cells, and the iris tissue markedly infiltrated, there is *fibrinous iritis;* if with an exudation rich in cells and fibrin there is extensive infiltration of the iris tissue, with mononuclear and multinuclear leukocytes, there is *purulent iritis* (Ginsberg).

Treatment.—The description of the treatment is reserved for the subsequent sections devoted to the particular consideration of the various types of iritis which follow.

Iritis and Its Types

In the acute form of the disease the salient symptoms of iritis already recorded (see page 337) are present. Not only may the ordinary attachments form between the iris and the capsule of the lens, but a plastic exudation may cover the pupil space with a false membrane, and the adhesions between the iris and the lens-capsule may be unusually firm and unyielding. To this form of iritis the descriptive term *plastic* is sometimes applied. In some cases a gelatin-like mass is deposited in the anterior chamber, and its appearance has been compared to that of a dislocated lens in the same position (*fibrinous* or *spongy iritis* (see also page 776). Sometimes if synechiæ are elaborate the intra-ocular tension in iritis or iridocyclitis (page 367) may rise and may be associated with increased haze in the cornea, but haziness of the cornea, often present (page 338) is not of itself proof that ocular pressure is increased. In various forms iritis is due to—

1. **Syphilis—Syphilitic Iritis.**—The percentage of patients with syphilis who acquire iritis during the course of the disease varies from 0.42 to 5.37, according to the different authorities, but among cases of iritis, syphilis has been found to be the cause in from 30 to 60 per cent. (Alexander). Groenouw among 2020 patients with syphilitic ocular disorders found the percentage of iritis to be 44.7. Iritis develops not only in untreated syphilitic subjects, but also in those who are under treatment and occasionally has arisen after an injection of salvarsan

("Iridorezidive" Ternlink, quoted by Igersheimer). Trauma of the eye, a blow, for example, may in the syphilitic individual be the exciting cause of an iritis (Igersheimer). Briefly, syphilis is usually regarded as the most common cause of iritis, which may appear between the second and the ninth month after the initial lesion, or may be delayed until the eighteenth month. Occasionally it arises at a very late period in syphilis—that is to say, during the period of so-called tertiary manifestations, either as a primary iritis, as a relapse, or in one of the forms presently to be described. The lesions are due to the influence of syphilitic virus, that is, to the lodgment and activities of the Spirochæta pallida, which has been found in the aqueous humor of an eye with acute syphilitic iritis (Zur Nedden, Stephenson).

The clinical manifestations of syphilitic iritis vary. They may be those of ordinary acute iritis with lesions which of themselves do not justify the diagnosis of syphilis, and syphilis as the etiologic factor can be established only by the history of the case, by the therapeutic test, or by the serum reaction of Wassermann. According to Krückmann, an early manifestation of syphilis in the iris, which may appear in the sixth week after primary infection, is *roseola*, characterized by overfilling of superficially placed vessel loops, which arise and disappear quickly. The color is bright red in contrast to the copper tint of the skin eruption. Roseola may in later stages be the forerunner of papules.

Localized sphincter lesions always suggest the influence of syphilis, and with the development of papules a form of iritis appears which yields characteristic, if not pathognomonic, signs of its origin. In the inflamed iris there appear one or more yellowish, reddish-yellow, or reddish-brown nodules, varying in size from a hemp-seed to a small pea, situated at the pupillary or ciliary border, or occasionally between the two in the iris tissue, although Fuchs maintains that they do not arise in the midbreadth of the iris. They vary in number from one to four, the intervening tissue being comparatively unaffected, and belong, in spite of their resemblance to gummas, with which at one time they were confused, to a comparatively early period of syphilis—that is, to the period indicated in the previous paragraph. Under the influence of treatment they are gradually absorbed without leaving very marked scars, although a certain amount of atrophy of the iris tissue on close examination will be found marking their former situation. This form of iritis is sometimes called *true syphilitic iritis* or *syphilitic parenchymatous iritis*. It is also known under the terms *iritis papulosa*, or *condylomatosa*.[1] These names have originated because the small nodules in the iris have been compared to papular syphilids and condylomata, inasmuch as they belong in the same stage of syphilis to which these manifestations pertain. They clearly differentiate themselves from gumma, not only by the date of their appearance, but because they do not caseate or break down and suppurate.

[1] Krückmann objects to the term "iritis condylomatosa" because there is no accurate comparison to be made between condyloma of the skin and the papules of the iris in syphilis.

Even where these distinct nodules are not present in syphilitic iritis, careful examination of the iris will frequently show localized discolored swellings in the edematous iris tissue, and usually broad and thick synechiæ, formed by a union of the iris tissue with lens-capsule, and not merely an adhesion of the posterior epithelium, and, as Fuchs has shown, microscopic investigation indicates that these nodules are always present, but are sometimes so small that they have not sufficiently elevated the iris tissue to reveal their presence to ordinary clinical methods of examination. To this form of iritis the name *syphilitic* or *luetic fibrinous iritis* is sometimes given.

The disposition and character of the papules which develop on the iris as the result of syphilis have been particularly studied by Krückmann, who among the early varieties describes superficial and deep-seated *small iris-syphilids*, the chief situation of the latter being in the vessel system of the sphincter and its immediate neighborhood, and *medium-sized papules* which develop under the anterior stroma layer.

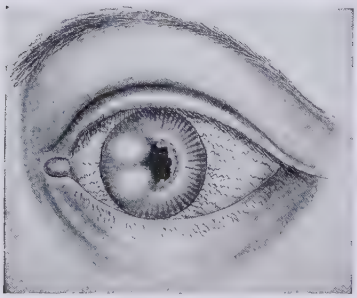


Fig. 156.—Papules in syphilitic iritis (from a patient in the Philadelphia General Hospital).

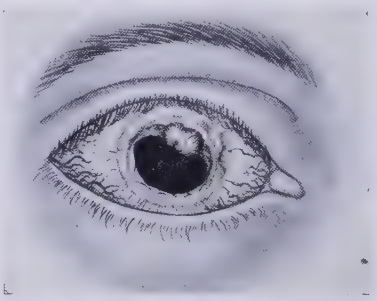


Fig. 157.—True syphilitic iritis with long nodes in iris; about seventh month of the disease, pupil partially dilated (patient in the University Hospital).

The papules of an early stage of syphilis often have a reddish color, while those of later periods of the disease are yellow or grayish-yellow and more decidedly circumscribed, owing to the absence of edema. A rare manifestation is the eruption of the papules in a group formation.

Gummatous iritis or, more properly, *gumma of the iris* occurs, appear, according to Alexander, almost constantly at the ciliary border. The lesion is solitary, of the size of a pea or small nut, and grows toward the ciliary body, disappearing through fatty degeneration, leaving behind a permanent scar, or *atrophy of the iris*. Such a manifestation, strictly localized in the iris, is extremely rare. It appears, if at all, in the so-called tertiary period of syphilis, or that period in which gummas in other organs are found (see also page 368).

In syphilitic iritis both eyes may be attacked, one a little later than its fellow, approximately in 25 per cent. of the cases; occasionally the onset is simultaneous. The course usually is acute, and after thorough cure relapses are not common. Sometimes the disease assumes a sub-

acute type, or may be so prolonged in its course and complications as to justify the term *chronic iritis.*

Acute iritis of the so-called plastic type is rare in newborn infants of syphilitic heritage, but has been described in children with *inherited syphilis,* from the second to the fifteenth month. Acute iritis in children in the first months of life, and also in later childhood years, usually is the result of hereditary syphilis. A later manifestation appears in the form of an iridocyclitis of the so-called serous type, the involvement of the entire uveal tract being evident by the manifestations of the signs of uveitis, which are elsewhere described.

Treatment.—The most important local drug in this as in other forms of iritis is atropin sulphate, gr. iv to f℥j (0.26 gm. to 30 c.c.), several drops of the solution to be instilled in the conjunctival culdesac every three or four hours. Mydriasis should be maintained until all ciliary irritation has subsided and during the period of changes in the refractive power of the eye (see page 338). Should the iris stubbornly resist the action of a solution of this mydriatic its strength may be increased (1–1.5 per cent.).

Pain is relieved and at the same time congestion is diminished, thus rendering the mydriatic action of the atropin more certain, by leeching the temple—one to three Swedish leeches being applied near the line of the hair, or blood is drawn by an artificial leech. In the absence of a regular heurteloup, this may be accomplished by making an incision in the temple with a scalpel and using a small cupping-glass, to which a piston is attached for exhausting the air. Should atropin not be tolerated, hyoscyamin, scopolamin, or duboisin may be substituted (see page 127).

The constant use of atropin leads to disagreeable dryness of the throat. This may be obviated in part by compressing the tear-duct after each application. It may be relieved by giving the patient a gargle made of equal parts of iced water and a strong decoction of coffee.

Pain is further relieved by the application of moist or dry heat; the latter is best made by means of cotton batting which is held before a fire and then laid upon the affected eye, to be replaced by a freshly heated mass as soon as cooling occurs, or with a Japanese stove or hot box. Moist hot applications are more efficient if a pad of surgical gauze is steeped in the following solution: Acetate of lead, ʒj (3.9 gm.); powdered opium, ʒss (15.5 gm.); boiling water, Oj (473.11 c.c.) (Randolph). Dionin in 5 to 10 per cent. solution is valuable on account of its lymphagogue and analgesic action, which is increased by the addition of a 2 per cent. solution of holocain. With the use of *high-frequency* currents for the relief of the pain of iritis the author has had no experience.

The best constitutional treatment is some form of mercury, either the protiodid, blue mass, or calomel, given as in syphilis generally, just short of the point of salivation, and continued for many weeks even after all acute symptoms have subsided. Inunctions of unguentum hydrargyrum are advantageously employed, preceded by a hot bath or

diaphoresis with the aid of a hot chamber. If inunctions are properly given they represent a most satisfactory method of administering mercury, and usually ʒj (3.9 gm.) of the ointment may be daily rubbed into the skin. Hypodermic or, rather, intramuscular injections of mercury, particularly mercuric chlorid or salicylate of mercury (of mercuric chlorid the dose may be from $\frac{1}{16}$ to $\frac{1}{32}$ grain—0.00405–0.002 gm.; of salicylate of mercury, $\frac{1}{2}$ grain—0.0324 gm.), are strongly advocated by some surgeons.

In syphilitic iritis *salvarsan* produces the most favorable results, and under its influence the lesions disappear with astonishing rapidity. If the Wassermann test is positive, other things being equal, a dose of 0.6 gram should be given intravenously (in women 0.4 gram is usually sufficient). At the expiration of three weeks, the Wassermann test being still positive, this dose may be repeated. During the intervals mercury or iodid of potassium should be administered.

Clinical trial of *neosalvarsan* indicates that it equals *salvarsan* in efficiency and that it is less toxic. In arsenic content 0.6 gram of salvarsan is equivalent to 0.9 gram of neosalvarsan. The injections may be given at comparatively frequent intervals, the dose being regulated according to circumstances and effects, as often as once in ten days or two weeks. At the present time in this country *arsphenamin* and *neo-arsphenamin* are used in place of the formerly employed salvarsan and neosalvarsan. While the American and German products are practically equivalent in so far as their dosage is concerned, B. A. Thomas suggests the treatment should begin in the adult female with 0.4 gram, and in the adult male with 0.5 gram of arsphenamin, which, if well tolerated, may after one or two injections be increased to 0.6 gram. If neo-arsphenamin is selected the first dose for a female should be 0.6 gram and for a male 0.75 gram, to be increased as before to 0.9 gram.

In old syphilitics with much cachexia, in whom a plastic iritis improperly treated in the early period has relapsed, it is not always wise or possible to induce active mercurialization. For them bichlorid combined with the tincture of iron is a suitable remedy. Subconjunctival injections of bichlorid of mercury (2 to 4 drops of a 1 : 2000 solution) are efficient, but painful. Acoin added to the injection relieves the pain. In place of the bichlorid solution one of cyanid of mercury (1 : 5000) is advised by Darier. Equally good results are obtained with 5- to 15-minim (0.3–0.92 c.c.) injections of physiologic salt solution if there is not too much circulatory stasis. The injections may be given every second or third day, and should be followed by light massage of the eyeball. Usually they are not necessary if active constitutional treatment (arsphenamin, mercury) has been carefully carried out.

2. **Rheumatism—Rheumatic Iritis.**—According to Krückmann, rheumatic iritis is apt to begin with conjunctival hyperemia or a nonbacterial conjunctivitis; at first, in some cases, there may be mydriasis with preservation of the pupil reflexes and congestion of some of the radially placed larger iris vessels, followed by a sudden increase of the

iris injection and the appearance of pericorneal injection, pupil immobility, fibrous exudation into the superficial stroma layer, and fine deposits on the posterior surface of the cornea. The vitreous remains clear.

The association of iritis with *acute rheumatism* (acute rheumatic fever), however, must be exceedingly uncommon, if it ever occurs. Indeed, as Krückmann maintains, the so-called rheumatic iritis must be sharply separated from those iritic involvements which follow or accompany acute joint rheumatism, and which depend upon metastasis of staphylococci or streptococci proceeding from purulent processes in the joints. Paine and Poynton have isolated a diplococcus which they regard as the specific cause of rheumatic fever, and with which experimentally they were able to produce an iridocyclitis which was regarded as a true rheumatic iridocyclitis. In so far as acute rheumatism is concerned these observations have been confirmed by Rosenow, whose investigations also indicate that muscular rheumatism (see below) may be due to a closely related streptococcus.

Because the relation of rheumatism to the development of iritis is a vague one, and because the general "rheumatic" conditions—myalgia, joint lesions, etc.—may be toxic in origin, T. Harrison Butler suggests that the ocular manifestation should be denominated *autotoxemic* or *toxemic iritis*, and that the term "rheumatic" should be abandoned. There is no doubt that many cases of iritis, which formerly were characterized as "rheumatic" are due to gonorrhrea and to other focal infections.

3. **Autotoxemic Iritis (Iritis with Disorders of Nutrition or Constitutional Disorders).**—Notably between the ages of twenty and fifty, but also at later periods of life, either coincident or not with affections which are usually classified as chronic rheumatism, chronic joint rheumatism, or muscular rheumatism—*i. e.*, myalgia manifesting itself as lumbago, pleurodynia, or pain in various groups of muscles (usually classified by systematic writers as "constitutional diseases" or "diseases of nutrition")—iritis is not uncommon. In Butler's statistics this type of iritis or iridocyclitis constitutes 6 per cent. It varies considerably in the aggressiveness of its symptoms. Not uncommonly these are severe, with much pericorneal injection, acute pain, greater usually than in syphilitic cases, and tenderness of the globe. Frequently only one eye is affected; the inflammation rarely is simultaneously symmetric. The second iris may be affected in like manner after a longer or shorter interval. From the subjects of this form of iritis, if none of the various types of polyarthritis or myalgia is present, a history of such an affection can usually be obtained; sometimes the history develops the fact that the patient has suffered from sciatica, or crural, musculospinal, and other forms of neuritis. In many cases areas of focal infection (teeth, tonsils, sinuses, intestinal tract, etc.) are in evidence and are related etiologically to the iritis and the chronic rheumatism and myalgia, each being manifestations of the toxemia thus produced.

Relapses are frequent, in this particular differing from syphilitic plastic iritis, and a patient once having had an attack of this type of iritis (often in its manifestations an iridocyclitis) is liable at intervals of months or even years again to be attacked. If treatment is begun early, even in recurring attacks, perfect cure may be expected.

The frequent relapses of some varieties of the affection have given rise to the term *recurrent iritis*. This form of iritis has been noted in association with ictero-hemorrhagic spirochetosis; recurrent iritis with dermatitis exfoliativa has been reported (S. Gifford).

A form of iritis exists, aptly called *quiet iritis* (Hutchinson), in which there is no pain or ciliary congestion; it is practically non-adhesive, the only subjective symptom being the progressive dimness of vision, which leads to its discovery, and which is associated with so-called chronic rheumatism or inherited arthritic tendency or the uratic diathesis in a majority of cases, but which may also depend upon syphilis. A variety of quiet iritis in which the lesions are said to be confined to the posterior layer of the iris has been described by Grandclément under the name "Uvéite irienne." It occurs usually in women during the period of uterine activity; frequently its subjects are anemic. Grandclément failed to associate it with any local or general malady. One variety of iritis, moderate in its manifestations, although it may be associated with small hemorrhage in the iris tissue, is due to arteriosclerosis and occurs in middle-aged or elderly persons (Michel). Iritis in the course of chronic nephritis is also occasionally encountered.

A severe and sometimes destructive form of iritis may accompany *arthritis deformans*. There is a variety of iritis characterized by warty-looking translucent excrescences at the pupil margin, generally occurring in women, to which Doyne gave the name *guttate iritis*. (Compare this section with pages 361–367).

With two constitutional diseases or disorders of nutrition iritis or iridocyclitis is not infrequently associated, namely, gout and diabetes.

Gouty iritis, as it is usually called, occurs in the subjects of gout, irregular gout (often referred to with questionable accuracy as gouty, lithemic, or uric acid diathesis), and in the members of gouty families. It probably depends *indirectly* upon the defective nitrogen metabolism which underlies gout, although this origin is not accepted by some authors. It tends to relapse and to attack one eye at a time; the superficial layers of the iris are especially affected. It may precede a gouty attack elsewhere in the body. A form of iritis, insidious in character and destructive in tendency, almost invariably associated with disease of the vitreous, occasionally occurs in children of gouty parents. These children, according to the late Mr. Hutchinson, have a peculiar squareness of build, heavy features, florid complexions, and feebleness of circulation in the extremities.

Less frequently iritis (it may be purulent), sometimes associated with fibrous deposits in the pupil space and with hemorrhage in the anterior chamber, develops in the subjects of diabetes; it is ordinarily described as *diabetic iritis*, but more properly should be considered as

an iritis which occurs in a diabetic subject. Its frequency among diabetics has varied from 1.5 to 5 per cent.

For an inflammation of iris associated with gout, diabetes, and hypothyroidism, Duane proposes the name *metabolic iritis.*

Treatment.—The use of atropin in the manner already described is of paramount importance. For it scopolamin, gr. ij to f℥j (0.13 gm. to 30 c.c.), may be substituted, or the two drugs may be combined. Leeches and moist and dry heat help to relieve the pain, and, at the proper stage, subconjunctival injections of salt solution. Much comfort often results from the administration at night of $\frac{1}{100}$ grain (0.00065 gm.) of hyoscin, but morphin or codein, if given at all, must be administered with great caution lest the patient form a drug habit. Rubbing the brow with an ointment of mercury and belladonna is of some service. Dionin (5 per cent.) and holocain (2 per cent.) act well in relieving pain.

Much reliance may be placed upon salicylic acid, salicylate of sodium, salicylate of strontium, and acetyl-salicylic acid (aspirin); of these remedies, salicylate of sodium is the best; indeed, it relieves the pain of any form of iritis. It should be exhibited in full doses, 60 to 80 grains (3.9–5.2 gm.) during the first twenty-four hours, and afterward the amount gradually lessened.

The tendency to recurrence requires preventive treatment in the form of regulated diet, the use of mineral waters, and proper attention to change of clothing, according to the vicissitudes of the climate. A course of treatment at some establishment connected with the various medicinal springs is of great benefit. In all forms of iritis, especially in the autotoxemic types, the nasopharynx, the tonsils, the teeth, the accessory sinuses, and the buccal mucous membrane should be carefully examined for focal infection, which may be the cause of the toxemia of which the patient is the subject as well as of the iritis (see also page 366). Intestinal sepsis may be present and must be corrected.

If the iritis assumes a chronic type, or if there has been exudation of lymph or involvement of the ciliary body, mercury and iodid of potassium may be exhibited. After the inflammatory signs of iritis have thoroughly subsided and the eye is quiet, the refractive error should be thoroughly corrected and the glasses worn constantly, because there is no doubt that this plan of treatment distinctly checks the tendency to relapse.

Should gout be determined to be the underlying cause, the usual treatment of this affection—dietetic and medicinal—is indicated, in addition to the local measures. Medicinal springs treatment is especially valuable. The subjects of diabetes must be treated on general principles, by regulation of the diet, etc.; it is probable that under proper conditions *insulin* may serve a valuable purpose.

4. **Gonorrhea—Gonorrheal Iritis (Gonorrheal=rheumatic Iritis).**—This is a form of *focal iritis* (often a severe iridocyclitis), chiefly plastic in character, which does not necessarily coincide with or immediately follow the gonorrheal attack; an arthritis of the knee, or of the ankle,

intervenes; sometimes arthritis and iritis occur at the same time, and the iritis may precede the arthritis. Brailey has seen it assume a gelatinous type. This disease is much more common than has usually been supposed, and there may be a long interval between the acquisition of the gonorrhea and of the iritis—frequently several years and even as long as thirty years. The blood should be examined for a complement-fixation reaction, and the posterior urethra for gonococci. It should be remembered that an ordinary plastic iritis in a person with syphilis may be due to a gonorrhea from which he has also suffered. Chronic forms of gonorrheal iritis resemble chronic endogenous uveitis (see page 361) and may be associated with tuberculosis (v. Herrenschwand). In many instances so-called rheumatic iritis is really gonorrheal in origin; indeed, William Lang believes that the gonococcus is the most frequent cause of plastic iritis. The affection is due to the influence of the gonococci and their toxins on the iris. The presence of these micro-organisms in the anterior chamber has been demonstrated (Sidler-Huguenin). The disease is attended by severe pain, in addition to the usual symptoms of iritis, and its chief manifestations are in the superficial layers of the iris. It may relapse with each new attack of gonorrhea.

Treatment.—The local use of atropin, etc., is indicated. If the urethra is inflamed, this must receive attention. Iodid of potassium may be tried, and mercury, if there is much exudation. Relief follows profuse sweats by means of pilocarpin given hypodermically or with the aid of an ordinary hot chamber or cabinet; indeed, these remedies are of great value in other varieties of stubborn iritis. Subconjunctival injections of salt and of cyanid of mercury have been advocated, and dionin in the usual manner should be employed. Excellent results, in the author's experience, follow the administration of gonococcic vaccine (Neisser bacterin). Large doses are tolerated: 50,000,000 to 100,000,000 organisms may be injected at intervals of three to seven days. Even larger doses are advocated by William Lang (200,000,000 to 500,000,000 at intervals of a week). John Weeks, however, thinks smaller doses (2,500,000 to 50,000,000) produce equally good results.

Iritis Secondary to Mucous Membrane and Focal Infection (Focal Iritis).—Iritis (often iridocyclitis or uveitis) caused by infection of mucous membranes is of frequent occurrence and has been referred to in the discussion of autotoxemic iritis (page 347). The primary source of such infection most frequently is a chronic septic process in the mouth (pyorrhœa alveolaris), in the teeth (tooth-root abscesses), in the tonsil, in the nasopharynx, in the accessory nasal sinuses, in the middle ear, in the stomach and intestines, in the gall-bladder and appendix, in the urethra (see page 349), in the uterine cavity, the prostate, the seminal vesicles, the bladder, and in the skin (boils, furuncles, etc.). W. L. Benedict, reporting the results of researches in the Mayo Clinic, states that cultures from dental abscesses revealed streptococci, staphylococci, and Streptococcus viridans. Animal experimentation demonstrates that the last-named micro-organism is the most potent one, and is responsible for an iritis which from the pathologic stand-

point may be regarded as a myositis. The anterior layers of the iris are chiefly involved according to Sanford Gifford's studies.

Treatment.—In all cases of iritis and iridocyclitis the regions named must be carefully searched for disease and persistently treated if it is present. The usual local ocular treatment is indicated. Vaccine therapy may be efficient. Both autogenous and stock (mixed) vaccines have been largely employed, and by some surgeons are highly commended. Autogenous vaccines are preferable, and in the author's experience, but almost entirely in focal iritis or iridocyclitis due to urethral and prostatic infection (not always gonorrheal in character), they have acted or seem to have acted favorably. With parenteral injections of sterilized milk (page 284) in the treatment of iritis the author has had no experience; they have been recommended. (For additional information see pages 366, 367.)

In malaria a *periodic iritis* with hypopyon has been described, and somewhat analogous to this is another periodic iritis, or iridocyclitis, which has been observed prior to each menstrual period (*iritis catamenalis*), perhaps due to abnormalities in the uterine discharge. Fuchs reports severe iridocyclitis in association with general alopecia. A relation between nephritis and iritis has been described. Sometimes plastic iritis of moderate grade is found in elderly persons for which no cause can be found; to these types of iritis formerly the name "idiopathic" was applied.

Tubercle of the Iris (*Tuberculous Iritis*).—In a certain number of persons, usually between the ages of five and twenty-five, small, grayish-red or yellowish nodules develop at the margin of the pupil or at its ciliary border, bearing great similarity in their external appearance to miliary growths (*disseminated miliary tubercle of the iris*). The nodules are usually 2 to 3 mm. in diameter, and may be situated close to the anterior surface of the iris or deep in its stroma. Two terminations have been observed: the growths may develop slowly and finally may be absorbed and disappear, posterior synechiæ remaining at their points of origin (*attenuated tubercle of iris*, Leber); or successive developments of new nodules may lead to a plastic inflammation of the iris and ciliary body, and involvement of the cornea (*keratitis punctata*), and cause perforation at the corneoscleral junction and shrinking of the eyeball. In these circumstances tubercle of the iris appears in the form of an iridocyclitis (uveitis). In some cases of tuberculous iritis the nodules are ill-defined, being situated within the inflamed and thickened iris.

Tubercle of the iris also occurs in a *confluent* or *conglomerate* form, a yellowish nodule growing from the periphery of the iris, covered, it may be, with smaller bodies. The tendency of this growth is to increase, to perforate the eye, and to cause a general dissemination of tubercle.

The average age of persons affected with primary tuberculosis of the iris is twelve years; one or both eyes may be affected, more commonly the former. Although to ordinary clinical inspection the patients may present no other signs of tuberculosis, this, and in a fatal form, may be a sequence. But the iris lesion is always a secondary infection,

that is, there is a tuberculous focus elsewhere in the body; it may be only a lymphatic gland. Sometimes the affection of the iris is secondary to the general disease. Bacilli and giant-cells may be found in these growths, proving their true nature. For diagnostic purposes Koch's old tuberculin may be used in gradually increasing doses, beginning with 1 mg. and with two-day intervals, increasing to 5 mg. Prior to its use the patient should receive a thorough general examination by an internist in order to ascertain whether there are any contraindications for test. Exceptionally the inoculation of a rabbit's or guinea-pig's anterior chamber with a fragment of the suspected tissue may perhaps be justified, but the danger of such a procedure has been explained.

Treatment.—Removal of a tubercle of the iris by an iridectomy is practically always unsuccessful. It should not, as a therapeutic measure, be performed; it might readily be responsible for the dissemination of tuberculosis. Hence, if the disease is attacked from the operative standpoint, enucleation has been recommended. Before radical surgical procedures are adopted there should be a thorough trial of the therapeutic value of tuberculin. A bouillon filtrate of tuberculin obtained from the Saranac Laboratory, with the initial dose of 0.0001 mg., is recommended by G. S. Derby.[1] The author has used with satisfaction tuberculin "old." The five dilutions contain respectively 1 : 1000 mg., 1 : 100 mg., 1 : 10 mg., 1 mg., and 10 mg. The dose of each dilution is 2 mg., and is progressively increased until 20 minims are injected. Following this, the next series is begun. The author's results with tuberculin therapy, employing various preparations, have sometimes been satisfying; sometimes quite indifferent or negative. It is difficult in many instances to estimate its value. The introduction of iodoform into the anterior chamber has been tried. Phototherapy has been recommended (Seidel). With these methods of treatment the author has had no experience.

Scrofulous iritis occurs usually in children and young persons of scrofulous habit. In some respects it resembles inherited syphilitic iritis. Nodules of lardaceous appearance may also form. *Tuberculous iritis* is described on page 351.

Infectious Disease Iritis; Metastatic Iritis.—This is seen in association with recurrent fever, variola, pneumonia, pertussis, parotitis, tonsillitis, herpes zoster, cerebrospinal meningitis, influenza, dysentery, typhus and typhoid fever, and infectious icterus and a *purulent iritis*, as the result of embolism, occurs in the course of septicemia after puerperal fever and in pyemia. In variola white spots may appear in the iris (*vitiligo iridis* [Fuchs]). The iridocyclitis observed in association with dysentery may or may not be accompanied by articular disease (Morax).

[1] This initial of tuberculin injection, that is 0.0001 mg. may be repeated every four days, adding to each injection 0.0001 mg. until a dose of 0.001 mg. is reached; and then adding 0.001 mg. until 0.01 is reached; then every two weeks adding 0.1 mg. till 1 mg. is reached. This plan is the one followed in the Knapp Memorial Hospital and may be recommended.

An association of mumps and iridocyclitis has been known for a long time. There is a form of uveitis which has been especially described by Heerfordt as *febris uveo-parotidea*, that is, an implication of the uvea and parotid gland; the lymph-glands, lacrimal glands, and salivary glands may also be involved; facial palsy is a complication; sometimes the ocular disorder precedes the parotitis, sometimes the reverse; small nodules develop in the iris. Even if the glandular inflammation subsides, permanent ocular damage may be an ultimate result. George MacKay and A. Maitland Ramsay have reported similar cases; MacKay's patient had, in addition to uveoparotitis, iridocycloplegia; the eye ultimately entirely recovered. A *febrilis uveoparotitis*, exhibiting a chronic course, due to tuberculosis and yielding to tuberculin therapy, has been recorded (E. Schall).

The management of such cases depends upon general principles, the free use of quinin and stimulants being appropriate in purulent iritis.

Traumatic iritis occurs as the result of an injury, either accidentally inflicted or due to an operation—*e. g.*, cataract extraction. In this category are placed, also, those cases of iritis which follow discission of the lens and which depend upon swelling of the cortical material or toxins from the lens material and infection conveyed through the wound. Verhoeff and Lemoine have demonstrated that intra-ocular inflammation from lens matter occurs in the eyes of those individuals who are hypersensitive to lens protein, proved by the fact that it (the lens protein) produces a positive skin reaction. The inflammation thus produced is termed *endophthalmitis phacoanaphylactica;* it exhibits characteristic histologic changes (infiltration of iris with plasma cells; lymphoid cells on the posterior surface of the cornea, infiltration of the lens with pus cells). The iritis excited by foreign bodies—for instance, metallic particles—embedded in the iris has been ascribed to a chemic as well as to infective action.

Treatment.—The usual local measures are advisable. Iced compresses are advantageous, and the internal administration of mercury and the salicylates is indicated.

Sympathetic iritis (see page 370).

Secondary iritis, independently of the fact that in a certain sense all types of iritis are secondary, may depend upon exogenous sepsis, for example, that which results from an infected corneal wound (see Traumatic Iritis) or upon exogenous toxemia, for instance, that which proceeds from an infected corneal ulcer (see page 272).

Scleritis of the deep variety is often associated with iritis. More rarely the primary disease begins deep in the eye—*e. g.*, in detachment of the retina. The presence of intra-ocular tumors, vitreous exudations, or retinal hemorrhages may occasion a secondary iritis.

Serous Iritis.—At one time it was the custom to describe a form of iritis characterized by a serous or, more commonly, a seroplastic exudation, deepening of the anteror chamber, slight dilatation of the pupil, haziness of the cornea and aqueous humor, and a precipitate of

opaque dots upon the posterior surface of the cornea, generally arranged in a triangular manner, with the apex pointing upward, by the term *serous iritis*, or *serous iritis* and *keratitis punctata* (see page 362). Both of these terms are inappropriate, the one indicating purely a symptom of a disease and the other an unproved pathologic condition. For a full consideration of this matter see page 361.

Chronic Iritis.—Any type of iritis may assume an acute, subacute, or chronic course; if the last, no additional symptoms occur, but those ordinarily present are modified by the chronicity of the stages.

In addition to the chronic type of an ordinary iritis there remains to be described one which has received the name *plastic iridochoroiditis*, because of coexisting disease of the choroid and vitreous, leading to the formation of a secondary cataract. This disease occurs in adults, often

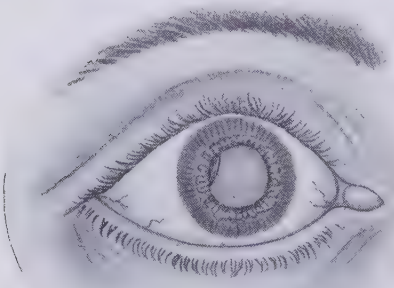


Fig. 158.—Cyst growing from posterior surface of the iris advancing into the anterior chamber (from a patient in the University Hospital).

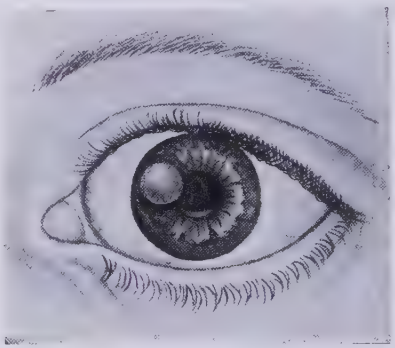


Fig. 159.—Cyst of the iris following traumatism (from a patient in the University Hospital).

without assignable cause, is symmetric, and proceeds steadily in a tendency destructive to the nutrition of the eye (see also page 364).

The *treatment* of the latter condition is unsatisfactory, alteratives, tonics, and operative measures often meeting with indifferent success.

Operative Treatment in Iritis.—Paracentesis of the cornea may be needed to reduce continued elevation of tension in some forms of iritis, and has been advised as a therapeutic measure in certain varieties of iridocyclitis. Should inflammation of the iris and hypopyon exist, the treatment already described (see page 281) is required.

Iridectomy is often recommended for the relief of recurrent iritis, or for an iritis which refuses to heal completely, some ciliary injection and irritability remaining. Those eyes which present the least change in the iris, in which the aqueous humor is clear and the tension is not subnormal, are most likely to yield a good result. Iridectomy in recurrent iritis does not insure the patient against future attacks, and repre-

sents a method of treatment which, in the author's experience, is often unsatisfactory except as it may be required to relieve increased intraocular tension.

In chronic iritis, circular posterior synechiæ and bulging of the iris are important indications for the operation. Determined rise of tension and threatening glaucoma, in any circumstances, furnish imperative reasons for its performance. According to the late Mr. Nettleship, keratitis punctata, chronic thickening of the iris with very extensive attachments, the existence of myopia, a tendency to spontaneous bleeding, and hypopyon render the operation less desirable; if the tension is below the normal, the operation may be followed by bleeding and shrinking of the eyeball; occasionally, even in these circumstances, excellent results are achieved. If, however, a chart of the visual field, sometimes obtainable only with a small point of light, indicates extensive deep disease, the chances of operative success are notably lessened and the operation may be contraindicated.

An iridectomy in these circumstances is performed to secure one or all of three results: (1) To prevent recurring attacks; (2) to re-establish communication between the anterior and posterior chambers of the eye, and thereby to enhance nutrition and check threatened glaucoma; (3) to improve vision by the substitution of an artificial pupil for one that has been occluded or excluded.

That portion of the iris should be selected for excision which is least changed and least bound down by adhesions.

Posterior synechiæ remaining after the acute symptoms of iritis have subsided have been regarded as a cause of relapse or recurrence, and, although this has not been proved (compare with page 341), several operations (not to be recommended in the author's opinion) have been devised for severing such attachments, to which the general term *corelysis* has been applied.

Tumors of the Iris.—**Cysts** having transparent, delicate walls lined with pavement epithelium (*serous cysts; retention cysts*) may be congenital or may develop in the iris as the result of an injury. They are due to closure of the mouth of an iris crypt and its distention by the retained fluid. Cysts formed by a separation of the two layers of the pigmented retinal epithelium at the back of the iris (*cysts of the retinal epithelium*) are due, according to Treacher Collins, to interference with the lymph-current of the iris. *Multiple cysts* may develop on the posterior surface of the iris (Pagenstecher, Fuchs, Wintersteiner). Cysts in this situation may have a brownish color and may be mistaken for a malignant tumor. A *parasitic cyst*—that is, one due to *cysticercus* in the iris—has been reported. Implantation of a cilium, or of superficial epithelium, in the anterior chamber may be the starting-point of an *epithelial, pearl-like tumor* (*pearl-cysts* or *cholesteatoma*), essentially cystic, with a lining of laminated epithelium and semisolid contents of degenerated epithelial cells and fat-globules (F. R. Cross and E. T. Collins). Traumatic cysts, which owe their origin to the intrusion through a wound of corneo-conjunctival epithelium, which proliferates,

are divided by J. Meller into *iris-cysts proper*, which are situated entirely within the iris tissue, *iris-chamber cysts*, which are situated partly in the iris and partly in the anterior chamber, and *wall-chamber cysts*, which are so situated that the iris forms only part of their boundary wall.

A cyst may be minute, or grow and fill the anterior chamber; both eyes may be affected, and some instances of multiple iris-cysts are on record. A cyst may cause iridochoroiditis by pressure. An attempt should be made to remove it through an incision, the growth and surrounding iris being seized, drawn out, and excised.

Sarcoma of the iris is rare as a primary growth. It has been well studied in this country by Veasey and by C. A. Wood and Brown Pusey. Iris sarcoma is more common in the latter half of life—that is, after thirty years—than at an earlier period, although a few cases have been reported in the first decade of life. Females are more often affected than males; the lower half of the iris is the primary seat of the growth in a large percentage of the cases; but ring sarcoma of the iris associated with involvement of the ciliary body may occur (T. M. Li). A few instances of bilateral iris sarcomas are on record. The first stage of the tumor's growth is slow, and may last for months and even years; in the second and later stages there is rapid increase in size, with pain, hemorrhage, etc., and, finally, rupture of the globe. Usually the tumor is pigmented; rarely a leukosarcoma of the iris develops. Histologically small round and small spindle cells are the predominating forms. The growth must be differentiated from melanoma, tubercle, and gumma. A few iris sarcomas have been successfully removed by iridectomy (Thorington); but Wood and Pusey are emphatic in their advice that the globe shall be enucleated as soon as the diagnosis is certainly established.

Melanoma of the iris is a dark tumor, developed from the pigment stroma of the iris, and although commonly passive and innocuous, is occasionally the precursor of sarcoma. Melanomas also occur at the pupillary margin of the iris, where they develop from the retinal pigmented cells.

Rare forms of iris tumor are vascular growths (nevi), leprosy nodules, and myomas. It is more than doubtful if primary carcinoma of the iris occurs; it may develop as a secondary growth, as also may glioma. *Metastatic carcinoma* of the iris secondary to breast carcinoma has been reported (Toulant, Proctor); also secondary to cancer of the lung and stomach (Usher).

Injuries of the Iris.—Wounds.—An incised wound limited to the iris does not necessarily produce serious results. Wounds, however, are rarely thus limited, but having penetrated the eyeball through the cornea or ciliary region, may cause sympathetic inflammation, or injure the lens and produce traumatic cataract.

In the first instance atropin, to secure physiologic rest of the iris, and a compressing bandage are indicated; in the other instances the extent and position of the wound determine the necessity for enucleation or for treatment suited to traumatic iritis.

Foreign Bodies.—A foreign body may penetrate the cornea and lodge in the iris, or, having partially penetrated the cornea, may be pushed through it in the efforts at dislodgment and become entangled in the iris. These foreign bodies include fragments of iron, steel, shrapnel, stone and wood, and detached cilia, which, passing in through a corneal wound, adhere to the iris. During the past war particles of unburnt cordite penetrating the cornea and lodging in the iris proved to be a serious ocular accident in many soldiers.

An opening is made with a broad needle or narrow keratome at the corneoscleral junction, eserin having been previously instilled, and a pair of forceps passed into the wound, with which the body is seized, or a small loop of platinum wire may be slipped beneath the fragment, by means of which it is withdrawn. If this is not possible, the piece of iris in which the substance is entangled may be drawn through the wound and excised. If the body is composed of steel or iron, it can be dislodged by means of a magnet.

Blows upon the eye may cause the following lesions:

Iridodialysis is a rupture of the ciliary attachment of the iris (ligamentum pectinatum). An opening is produced comparable to a false pupil; it may be detected by the red reflex which shines through the artificial aperture, usually somewhat semilunar shaped, situated in the periphery of the iris at the corneoscleral margin (Fig. 150). This may be quite small or it may involve more than half the circumference of the iris. The injury may produce other lesions—for example, cataract.

In a few instances reattachment of the ruptured fibers has taken place under the favoring influence of atropin, which should be vigorously instilled. An operation whereby the detached border is replaced and held in place by a stitch has been practised with success (Bulson). Ordinarily the lesion is permanent and, if small, occasions little trouble, although there may be diplopia. Pain, some dread of light, and hemorrhage into the anterior chamber are the immediate sequences of such an accident.

Rupture of the sphincter produces mydriasis and minute notchings of the pupil border. The not uncommon dilatation of the pupil (*traumatic mydriasis*) which follows a blow is always accompanied by such a lesion. The condition is not altered by treatment. Rupture of the continuity of the iris membrane by concussion is very rare.

Displacement of the iris occurs under three forms: (1) *Retroflexion*, or a folding back of a portion of the iris upon the ciliary processes, usually accompanied by a partial dislocation of the lens; (2) *anteversion*, or turning upon itself of the detached portion of the iris, so that the under or uveal surface is exposed; and (3) *aniridia*, or complete detachment of the iris from its insertion, so that it lies in the anterior chamber, or even under the conjunctiva. An injury severe enough to produce this condition usually is associated with other serious lesions of the remaining structures of the eye. In some cases, however, the aniridia is the sole injury and vision is not materially disturbed. In one patient under the author's care, the iris having been completely detached as

the result of the eye coming violently in contact with a sharp iron rod, vision is entirely normal with a correcting glass, blackened except at a central area corresponding in size to the pupil.

Although *alterations of the anterior chamber* and foreign bodies, exudations, and tumors within it are referred to in the various clinical



Fig. 160.—Traumatic iridodialysis and partial cataract; pupil under influence of a mydriatic.

sections, it is convenient to summarize in this place some of those which are conspicious. Physiologically, the anterior chamber is shallower in infancy and in old age than at other periods of life, and diminishes in its middle depth during the act of accommodation. Pathologic *deepening* of the anterior chamber occurs in luxation or absence of the lens,

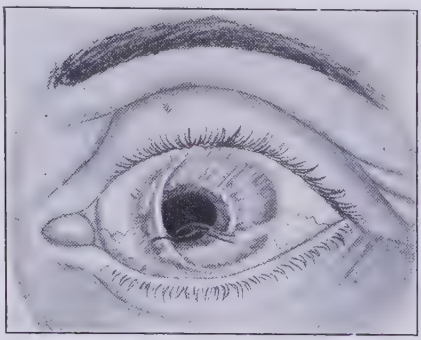


Fig. 161.—Cilia in the anterior chamber after wound of corneoscleral junction (from a patient in the Philadelphia General Hospital).

in some cases of cyclitis, and is present in conical cornea and certain forms of staphyloma and in buphthalmos. Pathologic *shallowing* of the anterior chamber occurs in chronic iritis with bulging forward of the iris, in glaucoma, and in the later stages of growths in the interior of the eye. Its depth is also lessened where there is diminution of the

secretion of aqueous humor, in long-standing inflammation of the uveal tract with detachment of the retina.

Alterations in its contents may consist in mere turbidity of the aqueous, as in iritis, keratitis punctata, and glaucoma, or there may be a collection of pus, several times referred to under the name of *hypopyon*, and commonly seen in sloughing ulcers of the cornea and purulent inflammations of the iris and ciliary body; also various types of exudation.

Finally, blood collects in the anterior chamber, a condition which receives the name *hyphemia*. This follows injury of the iris, and occurs in tumors of the eye, hemorrhagic glaucoma, and in severe forms of iritis and cyclitis. It is also seen in hemophilia and splenic leukemia (Sörger).

A foreign body penetrating the cornea may lodge upon the iris or fall into the anterior chamber. This may be a fragment of iron or steel or a particle of glass or any of the substances mentioned on page 327. Sometimes a cilium passing through a wound obtains entrance into the anterior chamber; if it remains long enough, it causes a cystic tumor (*implantation cyst*).

The two parasites may occupy the chamber, namely, *Cysticercus* and *Filaria sanguinis hominis*. In all these instances the intruder should be removed by an operation.

According to Parsons, endothelioma is the only primary growth of the angle of the anterior chamber. A tumor of this character, its cells being derived from the pectinate ligament, has been described by Hanke. Tubercle, gumma, sarcoma, and glioma occur as secondary deposits in this region. *Cysts*, derived from a congenital cystic growth of the ciliary epithelium, may develop in the anterior chamber (Holmes Spicer, R. A. Greeves).

CYCLITIS AND IRIDOCYCLITIS

Under the general term *cyclitis* are included various types of inflammation of the ciliary body. If the iris and ciliary body are associated in pathologic changes, the term *iridocyclitis* is employed (see also page 336).

The **symptoms** which justify the diagnosis of cyclitis or iridocyclitis are the following: Edema of the lid, injection of the circumcorneal or ciliary zone, neuralgic pain, and tenderness on pressure. Change in the aqueous humor, which grows turbid; grayish-brown points of exudation upon the posterior layer of the cornea, and at times hypopyon; exudation in the posterior chamber, attaching the under surface of the iris to the lens-capsule in a complete posterior synechia, the retraction thus produced causing a deepening of the anterior chamber; exudation into the vitreous causing opacities, especially in its anterior layers; and alterations in the tension of the globe, which may be increased or decreased. Various types of exudation in the anterior chamber, strands and membranes, are easily demonstrated by means of slit-lamp microscopy; also in a fair percentage of subacute and chronic iridocyclitis

"efflorescences" of the iris are visible. They occur at the pupillary pigmented margin; their finely granular surface contains pigment dust. Nodular lesions without pigment have been described (A. Vogt).

The *general symptoms* of pain, photophobia, lacrimation, etc., are present in the acute types of the disease, and vision is seriously impaired according to the amount of the exudation in the pupillary space and vitreous.

To those cases characterized by especially severe ciliary pain and marked pericorneal injection, dilatation of the veins of the iris and decided retraction of its periphery by reason of the plastic nature of the exudation in the ciliary body, the name *plastic cyclitis* is often given. The intra-ocular tension may be high or low, according to the grade of the inflammation and the character of the process. The disease may involve the choroid, and the vitreous may be filled with opacities. If the pain is comparatively slight and the pericorneal injection less marked, while deepening of the anterior chamber, primary slight dilatation of the pupil, turbidity of the aqueous, and decided precipitation of dots on the posterior surface of the cornea ("keratitis punctata") are conspicuous features, the descriptive term *serous cyclitis* is sometimes employed. With these phenomena fine vitreous opacities, inflammation of the iris and choroid, narrowing of the anterior chamber, increased tension, and secondary glaucoma may be associated.

Purulent cyclitis is characterized by intense ciliary pain, great pericorneal injection, and edema of the conjunctiva and the upper lid. The vitreous contains large opacities, and a noteworthy feature is the formation of hypopyon, which may disappear and reappear in a few days, its reappearance sometimes being signaled by a fresh exacerbation of intense pain. The iris and choroid commonly are included in the inflammation.

Pathology.—As already noted, systematic writers at one time were accustomed to divide cyclitis into *plastic*, *serous*, and *purulent* cyclitis. The objections to a classification of this character have been recorded in connection with iritis (see page 341). In general terms, inflammation of the ciliary body may be *acute*, *suppurative* or *purulent*, and *chronic*. In addition to infiltration of the iris and exudation in the anterior chamber, there are round-cell infiltration of the ciliary body, much more intense in the vascular ciliary processes than in the ciliary muscle, and lines of exudation into the posterior chamber and the vitreous. The retina, choroid, and nerve are also involved in a varying degree. Later the exudations organize and contract, producing atrophy of the ciliary body, proliferation of the pigment layers, and stretching of the processes toward the posterior pole of the lens. The exudations contain newly formed vessels, the lens becomes cataractous, and if the inflammation has been intense, the retina is detached and *atrophy* of the entire *eyeball* results. If the ultimate result of cyclitis is *phthisis bulbi*, the pathologic process has been a *chronic plastic cyclitis*, with an exudation rich in fibrin which has gradually changed into fibrous tissue.

Prognosis.—Cyclitis under vigorous treatment, begun early, may

subside; but the prognosis is always grave, because the disease is liable to orginate glaucoma, and in the purulent type, or in the plastic variety which has become purulent, tends to produce atrophy of the iris and choroid and, as described above, *phthisis bulbi.*

Shrunken balls of this character are often tender, readily become inflamed, and may produce sympathetic ophthalmitis; this is particularly true if the original inflammation has been a cyclitis of the plastic type, which probably remains in a *chronic* state.

Causes.—As already stated, primary and uncomplicated disease of the ciliary body is rare. The affection usually is part of a process which involves the choroid or iris, and, therefore, the same conditions and affections which cause iritis (see page 337) may originate cyclitis. A full consideration of the factors concerned with disease of this region will be found in the following section, and also in the section devoted to Diseases of the Iris.

Injuries are common causes of cyclitis, and the inflammation may follow operations upon the globe—*e. g.*, cataract extraction.

The **treatment** of cyclitis is practically identical with that of iritis, and, therefore, the directions need not be repeated.

UVEITIS[1]

In this disease the clinical as well as the pathologic manifestations are concerned with all of the component parts of the uveal tract. The inflammatory process either affects the whole tract or, first confined to one part of it, is liable to extend to one or both of the other two parts; sometimes it remains confined to the part first attacked. Although the disconnection of uveitis and iridocyclitis is in the main artificial, from the clinical standpoint it may be justified, and because of certain characteristic signs it has been accorded a separate description.

Causes.—In general terms, the causes of uveitis may be septic or toxic. A certain number of cases depend upon conditions already referred to in connection with iritis and iridocyclitis, namely, upon so-called constitutional diseases or disorders of nutrition—for example, chronic rheumatism, gout, arthritis deformans, and diabetes; on disturbances of the internal secretion—hyper- and hypothyroidism; on specific infectious diseases—influenza, syphilis, gonorrhea, tuberculosis, and specific fevers; on diseases of the blood—for instance, anemia; on auto-intoxications, particularly enterogenous auto-intoxication; on areas of sepsis (focal infections) in the pelvic region, urethra, bladder, the prostate and seminal vesicles, the intestines, gall-bladder, appendix, the middle ear, the rhinopharynx and accessory sinuses, gums (pyorrhea alveolaris, tooth-root abscess), the tonsils, the pharyngeal ring, and the skin (furuncles). (See also page 350.) Iritis and uveitis may develop after antityphoid inoculations.

[1] The terms *descemetitis, aquocapsulitis, keratitis punctata,* and *serous cyclitis,* sometimes employed as synonymous with uveitis, are inaccurate, and for reasons already stated should not be used in this respect.

Stock's investigations, both from the clinical and the experimental standpoint, indicate that tuberculosis is a frequent cause of *chronic uveitis*. Hypophyseal disease and chronic tuberculous disease of the uveal tract may be associated (Uhthoff). Hence, tests to determine the presence of tuberculosis should not be neglected in the study of this affection, and the serum reaction of Wassermann should be utilized to establish the presence or absence of syphilis as an etiologic factor. Occasionally uveitis develops at the time of the menopause; it may be associated with hypo-adrenalism. Undoubtedly gonorrhea is a cause of importance. Inasmuch as most of the inflammatory affections of the iris and ciliary body are due to microbic infection, there exist good grounds for believing that the proximate cause of all cases of *endogenous iridocyclitis* is the excretion by the ciliary body of the micro-organisms and their products (Stephenson). As already pointed out (see page 350), and quoting Stephen Mayou, it may be said that non-

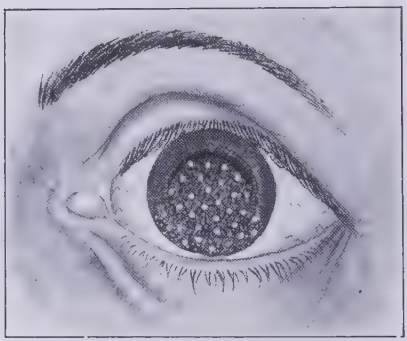


FIG. 162.—Uveitis, early stage; large dots irregularly placed (magnified and partly diagrammatic).

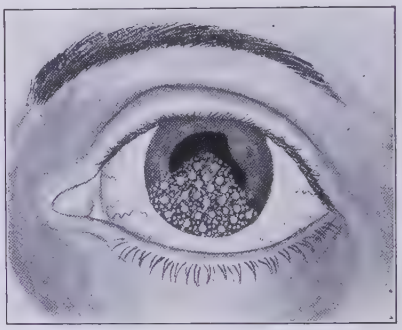


FIG. 163.—Uveitis; large dots on posterior corneal surface (magnified and partly diagrammatic).

suppurative inflammation of the uveal tract is often due to pyogenic organisms of diminished virulence, for example, *staphylococci*, derived from the distant foci of infection which have been named, and it has been attributed to *pneumococcus* and *streptococcus* recovered from feces, especially in chronic colitis (Browning) and to the *Bacterium coli* found in the urine (Lawson). In areas of dental infection the usual micro-organisms (staphylococci, etc.) are found as well as the *Streptococcus viridans* which is the potent micro-organism in these circumstances. The mechanism of focal infections in relation to the uveal tract inflammation from this source or indeed any source, accepting the theory of selective tissue affinity of certain bacteria, that is, elective localization, may be stated thus: bacteria find favorable opportunities for growth, multiplication, and entrance into the lymphatic streams not only by means of ulceration, but possibly by being carried in by migratory leukocytes acting as phagocytes. Having gained access to the blood-streams, they may be able to resist the bactericidal action of the blood

by reason of reduction in the resistance of the blood due to the primary infection. Subsequently they may locate in certain tissues, for instance, the uveal tract, either because they have developed a special affinity for these parts and find conditions favorable for their growth, or because the resistance of these parts is reduced by some other agency and thereby favors the localization of the germ (Kolmer).

Symptoms.—In large measure the symptoms of this condition have been described on pages 304 and 353, and in the paragraphs relating to the various types of cyclitis and iridocyclitis. In other words, the manifestations vary considerably, and one description does not apply to all types.

The following symptoms are often present, and when grouped together are characteristic: There are moderate deepening of the anterior chamber, at the beginning slight dilatation of the pupil (or, at least, an uncontracted pupil), haziness of the cornea and aqueous humor, and a precipitate of opaque dots upon the posterior elastic lamina of the cornea, generally arranged in a triangular manner with the apex pointing upward. There is slight pericorneal injection, and at first no great tendency to form synechiæ. It is not uncommon to find the tension somewhat higher than normal, at least in the earlier stages of the disease; later it diminishes. With the formation of posterior synechiæ, if they are at all extensive, secondary glaucoma may develop. The one fairly constant clinical sign, which in a certain sense is characteristic, is the manifestation called "punctate keratitis"—namely, a deposit of variously sized and colored dots, arranged usually in a triangular manner on the posterior layer of the cornea. In this connection, however, it should not be forgotten that this so-called descemetitis, in some form or other at least, is practically always present in all varieties of iridocyclitis, although not necessarily in the triangular-like manner which has just been described. The deposits on the posterior surface of the cornea may be fine and dust-like, or large and drop-like. To the last-named variety the term "mutton-fat" is often applied. The deeper layers of the cornea may be infiltrated (keratitis profunda; see also page 338), and the iris may contain thickenings and nodules, especially in the tuberculous types of the disease.

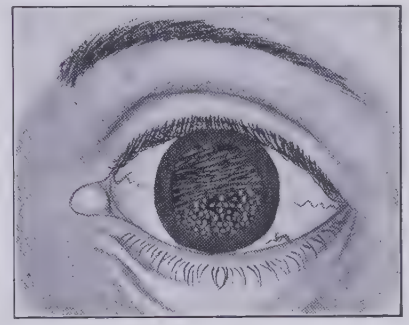


Fig. 164.—Uveitis, showing punctate deposits on cornea and cross-hatching (magnified).

Not only are the evidences of involvement of the iris and ciliary body present, but in most cases careful examination of the choroid reveals lesions in its tissues. They may be vague, and comprise only the so-called choroidal congestion, or appear far out in the periphery in patches of *acute choroiditis*, sometimes in large zonular areas of *acute*

plastic choroiditis, and sometimes so far forward that ophthalmoscopic examination does not detect their presence. The vitreous usually contains fine, floating opacities, or, in severe cases, coarse, web-like opacities, and occasionally hemorrhages. Visual field examination will often reveal, even in the absence of rise of tension, irregular contractions, and, not uncommonly in the earlier stages of the disease, *scotomas*. These scotomas T. Gray Clegg attributes to a toxic influence on the retinal elements at the macula and secondarily at the papillo-macular bundle of the optic nerve. According to Meller's investigations central and paracentral scotomas in this affection may be due to retrobulbar neuritis (Kleinsasser). The visual acuteness may be greatly decreased, owing to exudations in the vitreous and the deposits on the cornea, while in some of the milder cases it is scarcely reduced below the normal, and the patients are conscious only of ocular disease because of slight local discomfort and muscæ in their fields of vision.

There are so many manifestations of this disease that it is not practicable to attempt an exact classification. In general terms, it may be said that sometimes the signs are chiefly those of punctate keratitis or descemetitis; that a *senile* form of the disease is not uncommon, unassociated with acute symptoms or involvement of the iris, with only a few spots on the posterior surface of the cornea, and with a certain amount of flaky vitreous opacity, choroiditis in active manifestation being absent, although there may be a moderate degree of irregularity of the retinal circulation; that occasionally a marked descemetitis is the chief sign of a decided choroiditis or choroidoretinitis, often of obscure origin; that a *chronic uveitis* may develop involving the iris, less commonly the ciliary body, with or without punctate deposits in the cornea, insidious in character, more frequently encountered in young women than in men or in children; and that, finally, the disease may be recurrent and assume a type to which the author has ventured to give the name *malignant uveitis*, and which terminates in secondary glaucoma, cataract, and often in blindness.

With these cases of malignant uveitis may be described those to which the names *iridochoroiditis*, *cyclitis with disease of the vitreous and keratitis punctata*, and *chronic serous iridochoroiditis* have been given. They have been divided into two forms, according as the affection is primary in the iris or in the choroid. In the first instance, there are mild iritis, insignificant pain, and ciliary congestion, deepening of the anterior chamber, and spots on the posterior layer of the cornea; inflammation continues, relapses take place, exudation occurs behind the iris, while its pupillary margin is bound down so that the surface is irregularly bulged forward, and if the pupil is not too much occluded, the ophthalmoscope reveals many flocculi in the vitreous. The tension may now rise and the eye pass into secondary glaucoma.

In the other type the process passes from behind forward, beginning with patches of choroiditis, which increase in extent and depth; the nutrition of the vitreous is impaired and opacities form, the lens is altered and pushed forward, the iris becomes embedded in a plastic in-

flammation, with narrowing of the anterior chamber and a loss of vision. As the disease of the uveal tract continues, the lens becomes opaque, the eyeball softens, the retina may be detached, and, finally, phthisis bulbi occurs. In addition to the causes already mentioned, the affection, which is common in young adults, and usually symmetric, has been attributed to prolonged work, associated with loss of sleep and defective nutrition conditions, which, however, can be regarded only as contributory in the sense that they render the uveal tract more liable to a toxic influence.

Pathology.—The deposits on the posterior surface of the cornea, which are so conspicuous a symptom of this disease, and which have given rise to the name punctate keratitis, may be very fine or of medium size, and sometimes large and greasy looking, in which circumstances, as previously noted, they are known in England as "mutton-fat deposits." They are derived chiefly from the ciliary body, as Fuchs originally demonstrated, and also, in lesser degree, from the iris. An exudation of lymphocytes occurs upon the surface of the ciliary body, the lymphocytes pass into the anterior chamber and are precipitated on the posterior surface of the cornea (Fuchs). Their cyclitic origin has been strongly maintained by E. Treacher Collins, especially after his discovery of the so-called *glands of the ciliary body*. In other words, according to him, this disease may be regarded as primarily a catarrhal inflammation of these glands. Their secretion, he maintains, becomes augmented, causing increase in the aqueous humor and deepening of the anterior chamber. The aqueous is altered in character, contains leukocytes, pigment cells, and fibrin, and these formed elements gravitate and are deposited upon the lower portion of the posterior face of the cornea. Some authors—for example, Hill Griffith—have asserted that the dots on Descemet's membrane are formed in the choroid and set free in the vitreous, are carried by the nutrient currents of the eye and deposited on the back of the cornea. His view, as he himself points out, would necessitate the admission that the suspensory ligament is permeable to solid particles. Histologic investigation of the eyes affected with this disease indicates that in general terms there is a chronic cyclitis, in which the anatomic changes do not materially differ from those found in ordinary types of cyclitis. Groenouw has demonstrated round-celled infiltration of the iris, of the deeper layers of the corneal border and the ciliary body, with collections of round cells on the posterior layer of the cornea and on the ciliary processes. In his investigations the choroid, retina, and optic nerve were normal, but other observers have found them affected, as necessarily would be the case in so far as the choroid is concerned, where the ophthalmoscopic evidences of choroiditis are present. In later stages of chronic uveitis there is an overgrowth of the epithelium of the ciliary body. This epithelial hyperplasia, described by Fuchs, is, according to him, characteristic of some cases of chronic uveitis. Harms' investigation of the pathologic anatomy of chronic iridocyclitis with deposits on Descemet's membrane indicates that the inflammation of the uveal tract is limited largely to the anterior

segment of the eyeball, the deeper parts being much less affected. The pigment found in the deposits on the posterior surface of the cornea arises probably in part from degenerated red corpuscles, and largely from the uveal tract. In some grave types of iridocyclitis, leading to retinal detachment and atrophy of the eyeball, Fuchs found lesions resembling exogenous *endophthalmitis*, and believes they are caused by a virulent toxin or by bacteria conveyed by metastasis.

Treatment.—This depends upon the character of the disease or the type which it assumes. In the absence of increased intra-ocular tension, mydriatics are indicated, either atropin or scopolamin. With increased tension the mydriatic must be suspended and occasionally a miotic may be needed, or paracentesis of the anterior chamber may be performed. Dionin is of distinct advantage, and it may be combined with the atropin or eserin, according to indications. Pilocarpin diaphoresis is sometimes admirable in its effects. If for any reason the drug is not well borne, a similar physiologic action may be obtained by sweats induced in a Turkish bath, or with the aid of an ordinary cabinet. Subconjunctival injections are exceedingly valuable—either ordinary physiologic salt solution or cyanid of mercury. Mercury, preferably by inunction, even in non-specific cases, should be exhibited. It may also be given by the mouth in the form of protiodid. In syphilitic cases the indications for mercury are evident, and *salvarsan* and *neosalvarsan* or their equivalent, *arsphenamin* (see page 346), should be employed. Indeed, the remedy is of value in non-specific cases. Iodid of potassium, iodid of sodium, iodid of lithium, atophan, and sajodin are important alterative remedies, and in acute cases, especially those associated with great pain and decided cyclitis, full doses of salicylate of sodium or of aspirin render signal service. Naturally, the indications furnished by the probable etiologic factors must be given due consideration, and, therefore, iron, arsenic, bichlorid of mercury, syrup of hydriodic acid, and similar remedies should be administered. With atoxyl in this disease the author has had no experience, but he has seen marked benefit follow the administration of Donovan's solution. The administration of tuberculin in the manner already described (see page 352) may be followed by good results, and should certainly have full trial if the reaction to tuberculin is positive. But a critical examination of tuberculin therapy by Stock, while it indicated success in individual cases, showed a similar percentage of failure no matter which of the methods was employed. Mayou has successfully treated uveitis with *staphylococcic vaccine* where the staphylococcus has been obtained from the anterior chamber by paracentesis, and tests have demonstrated that neither tuberculosis nor syphilis is the etiologic factor. Indeed, *vaccine* prepared from cultures taken from the area of focal sepsis (from the tonsils, teeth, etc.) from which the active bacterial element is derived have been used with great advantage and are well worth trial (see also page 350). The author's investigations as well as those of Elschnig and other observers indicate that in a certain number of cases of uveitis, especially of an insidious and relapsing type, so-called enterogenous

auto-intoxication bears a relation to the disease, even if it is not a causal one in the sense that a toxin formed within the metabolism is the exciting agent, and treatment should include a carefully selected diet and intestinal antisepsis. Indeed, the association of intestinal sepsis with this disease is often an intimate one. Regulation of diet alone is not sufficient, although indigestible articles of food should be forbidden, especially sweets and carbohydrates. T. G. Dwyer has elaborated a method of treatment which consists in intestinal irrigations, notably of anhydro-sodium carbonate, and implanting in the *colon bacillus* and restoring the normal reaction of the intestinal contents. The Bulgarian bacillus internally is of value. The important relation of focal infection in the rhinopharynx, the tonsils, the teeth, and the accessory sinuses etc., to this affection has been referred to (page 361), and these regions should be thoroughly explored and treatment ordered according to the findings. The removal of infected tonsils, and the extraction or sterilization of septic teeth is of the greatest importance. The investigation of the teeth should always include an *x*-ray examination.

As Allen J. Smith and Barrett have found that the *Endamœba buccalis* is one of the factors in the production of pyorrhœa alveolaris, emetin has been used with success in relieving the oral sepsis, and thus indirectly may help in the cure of a uveitis caused by it.

After the acute symptoms have subsided, in the opinion of some authors massage of the eyeball is desirable, and galvanism has been advocated. S. Lewis Ziegler believes that electric treatment shortens the course of uveitis. He advises the application of the positive pole. In some types of severe uveitis the administration of thyroid extract has achieved encouraging results (Dunn, Bordley).

If as the result of this disease firm posterior synechiæ block the communication between the anterior and posterior chambers, this should be reopened by a broad peripheral iridectomy, which, if the lens is opaque, may be combined with its extraction. Even in eyes in which softening has begun, provided the field of vision still remains intact, good results sometimes follow a successful iridectomy. If at any time during the course of the disease rise of intra-ocular tension should develop and iridectomy should not be admissible, either paracentesis of the anterior chamber or posterior sclerotomy may be tried. Tapping the anterior chamber has been advised as a therapeutic measure, even if the tension is not elevated, and in some cases in which the author has put this expedient to the test it has been of benefit to the patients. The fluid from the chamber should be examined for the *Spirochæta pallida*, tubercle bacilli, and for pyogenic organisms. From the latter vaccines should be prepared as has already been advocated.

A few reports indicate that *radium* has served a useful purpose in relieving the pain and facilitating the absorption of inflammatory products in chronic uveitis (C. H. Williams); *x*-ray therapy (*actinotherapy*) has been employed; also phototherapy.

Injuries to the Ciliary Body.—The danger attending perforating wounds of the sclera has been described on page 327; this danger is

increased if the wound occurs in any portion of a zone, $\frac{1}{4}$ inch wide, surrounding the cornea, a region commonly called the "dangerous zone," after the late Mr. Nettleship's apt description. In addition to the damage inflicted by the wound the risk of acute and suppurative cyclitis and of sympathetic inflammation is present.

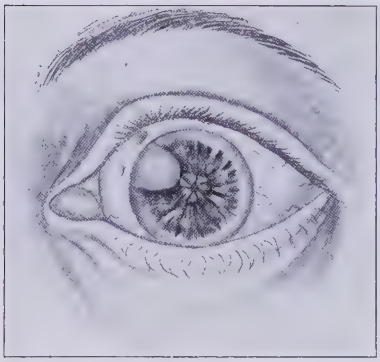


Fig. 165.—Gumma of iris and ciliary body.

Treatment.—After a penetrating wound in this region two courses are open to the surgeon—an attempt to save the eye, or immediate enucleation. If an attempt be made on the side of conservatism, the plan discussed under scleral wounds (see page 328) should be followed; if not, enucleation or one of its substitutes is indicated (see page 743).

Syphilis of the Ciliary Body.—Syphilitic affections of the ciliary body may exist either as a diffuse infiltration of granulation tissue and inflammatory exudation or as a tumor formation—that is, *syphiloma of the ciliary body* is present, to adopt the term advised by Ewetzky, who has

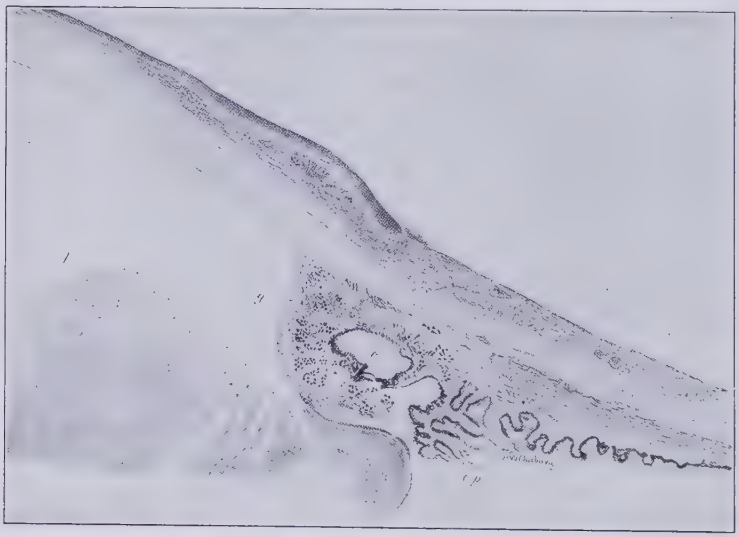


Fig. 166.—Microscopic section of gumma of iris and ciliary body (see Fig. 165) *l*, Remains of lens; *c.p*, atrophied ciliary processes; *g*, gummatous growth involving base of iris and ciliary body, containing in its center a cyst, *c*.

thoroughly studied this subject. According to him, the symptoms of syphiloma of the ciliary body manifest themselves chiefly in the form of a severe iridocyclitis, with hazy cornea, or as an intense parenchyma-

tous vascular opacity of this structure. Keratitis punctata may be a prominent symptom, and sometimes hypopyon is present. The tumor formation in the ciliary region may extend completely around the cornea, and perforation usually takes place through the sclera or into the anterior chamber. The color of the tumor is often yellow, and when caseous degeneration sets in there may appear in the anterior chamber the products of the degeneration of the tumor, which give rise to the appearance of hypopyon. The largest number of cases occur between the twentieth and the fortieth years of life, and more men than women are affected. Only rarely is inherited syphilis a cause of this condition. In those cases due to acquired syphilis, a large percentage has appeared in the early stage of the systemic affection.

A number of cases of *gumma of the ciliary body* have been recorded, but on pathologic grounds it is probably impossible to distinguish between papules and gummas, and, therefore, Ewetzky's term *syphiloma* is appropriate. Some writers describe *precocious gummas of the ciliary body* as early tertiary lesions, but Ewetzky does not believe that they should be separated into a special group. The condition must be differentiated from syphilitic growths of the conjunctiva which are movable; from syphiloma of the sclera, which is usually unassociated with iridocyclitis, and from melanotic sarcoma and tuberculosis of this region. The *treatment* does not differ from that which has been advised in connection with syphilitic diseases of the iris, and full doses of mercury and iodid of potassium are the most important therapeutic agents, especially if used in conjunction with salvarsan and neosalvarsan (see page 346).



FIG. 167.—Pigmented sarcoma of ciliary body protruding into the pupil space (from a patient in the University Hospital).

Tumors of the Ciliary Body.—Primary sarcoma of the ciliary body is a rare disease. Usually the growth is pigmented, although a few cases of leukosarcoma have been described. The sarcoma may not seriously impair the function of the eye in its earlier stages, when the tumor appears as a brown mass behind the iris, rarely in the angle of the anterior chamber. Later the growth exhibits the four stages which are common to all intra-ocular tumors. The tumor may be composed of round, spindle, or mixed cells, and, according to Groenouw, the prognosis is better than in sarcoma of the choroid. Sometimes sarcoma of the ciliary body assumes a flat and infiltrated character, to which the name *ring* or *annular sarcoma* has been given. This is a comparatively rare manifestation, only about 8 cases being on record (Alling and A. Knapp). The ciliary body may also be invaded by sarcoma from the choroid or iris.

Other growths which have been noted in this region are *epithelial hyperplasias* and *innocent tumors* of the *ciliary epithelium* (Fuchs), which, occurring in elderly persons, arise from the unpigmented layer at the summit of a ciliary process. An unusual pathologic condition found by Fuchs in an eyeball atrophic after iridocyclitis is a *neuroma of a ciliary nerve*. *Adenomas* have been described and, very rarely, *primary carcinoma*, *myoma*, and *myosarcoma*. For those tumors of the ciliary epithelium which represent the embryonic retina Fuchs proposes the name *diktyoma*. A tumor of this character has been described by Verhoeff with the title *teratoneuroma embryonale*. *Metastatic carcinoma*, *secondary glioma* and *hypernephroma* have been recorded.

As an extension of the disease from the iris or choroid, *tubercle of the ciliary body* may appear, and *leprosy nodules* have been reported, as have also a few *cysts*.

Senile degeneration of the ciliary body occurs, and *atrophic changes* in this structure may follow cyclitis. According to Parsons, *calcification* and *ossification* of the ciliary body itself are rare. Nearly always they are sequels of ossification in the choroid. A bony growth within the ciliary body has been reported (H. H. Brown).

SYMPATHETIC IRRITATION AND SYMPATHETIC INFLAMMATION OR OPHTHALMITIS

Sympathetic irritation is a functional disturbance in an eye previously sound caused by injury or disease of the fellow eye, probably due to irritation of the trigeminal branches. It presents a series of *symptoms*, comprising photophobia, lacrimation, blepharospasm, defective or impaired accommodation, lessened visual acuteness, inability to perform close work, neuralgic pain through the distribution of the supra-orbital nerve, tenderness on pressure over the ciliary region, photopsia, contraction of the field of vision (fatigued visual field), and hyperemia of the eye-ground.

Its *causes* are: Injuries of the eye, foreign bodies in the cornea and conjunctiva, keratitis, inflammations of the uveal tract, luxation of the crystalline lens, ill-fitting artificial eye, etc. The condition may recur and in this sense lasts for weeks and even months. It disappears promptly on removal of the exciting cause. It does not eventuate in sympathetic ophthalmia, although this disease is sometimes ushered in by irritative phenomena analogous to those just described, which rather unfortunately are also often denominated "sympathetic irritation."

This functional disturbance, that is, sympathetic irritation, as described, should be definitely separated from sympathetic ophthalmia as there is no sound evidence, as has been maintained by some writers, that its origin is infective and that the character of its manifestations is due to small or intermittent doses of the toxin.

Sympathetic Inflammation (*Sympathetic Ophthalmitis; Migratory Ophthalmia* [Deutschmann]; *Anaphylactic Uveitis* [Elschnig]).—

This is an iridocyclitis of one eye due to an affection of similar character in the fellow eye most often caused by injury. It is customary to describe the eye which is implicated as the result of disease or injury of its fellow as the *sympathizing eye*, and the one affected by the disease or injury which causes the irritation or inflammation as the *exciting eye*.

Conditions Producing Sympathetic Inflammation.—Generally one or other of the following conditions is present:

(1) Wounds, especially of the ciliary region which set up a traumatic iridocyclitis or uveitis, especially if associated with prolapse and incarceration of the underlying tissue or the capsule of the lens. The ciliary region is the zone previously described by the term borrowed from Mr. Nettleship as the "dangerous zone." Traumatisms probably cause over 80 per cent. of the cases of sympathetic inflammation. (2) Foreign bodies retained in the eye because of secondary infection or infection at the wound of entrance. It is improbable that a retained sterile chemically inert foreign body will cause sympathetic ophthalmia. (3) Perforating wounds or ulcers of the cornea in which the iris has become incarcerated, or scars involving the ciliary body. (4) Operations upon the eye—extraction of cataract, sclerotomy, iridodesis, iridectomy, discission, and reclination—followed by iridocyclitis. (5) Intra-ocular tumors, *e. g.*, sarcomas (Fuchs, Meller), if associated with iridocyclitis. (6) Ossification and calcification of the choroid and ciliary body and luxation of the lens; doubtless associated with iridocyclitis. (7) Incarceration of the stump of the optic nerve in scar tissue after the operation of enucleation; evisceration of the eye with remnants of uveal tissue remaining attached to the sclera and, in rare instances, implantation of an artificial vitreous in Tenon's capsule or in the scleral cup (Mules' operation).[1]

There is no sound evidence that herpes zoster ophthalmicus, glaucoma, symblepharon, intra-ocular cysticercus, or spontaneous inflammation of one eye can cause sympathetic ophthalmitis, although sympathetic irritation may arise in consequence of any of these conditions (Schirmer). Subconjunctival rupture of the sclera, with or without luxation of the lens, is said to have produced sympathetic ophthalmia. Meller has reported some cases of sympathetic ophthalmia, the exciting eye having acquired inflammation spontaneously, that is, without injury, but in each instance iridectomy had been performed on the eye originally attacked. Eyes which are, or have been, the subjects of purulent panophthalmitis very rarely produce sympathetic inflammation. This relationship has been recorded (Alt,[2] Schirmer, Ahlström,

[1] It is possible that in some of these cases enucleation or its substitutes may have been performed when the sympathetic inflammation was already present in its earliest stage.

[2] It is stated that eyes which are, or have been, the subjects of purulent panophthalmitis do not excite sympathetic ophthalmitis, and generally in suppuration of the cornea and its sequels and in panophthalmitis and the phthisis bulbi which it causes, this complication is not to be apprehended (Fuchs). Alt, however, in his analysis of more than 100 cases, found 13 eyes enucleated for sympathetic iridochoroiditis, the other having been lost by purulent panophthalmitis.

Zentmayer). According to Ruge, even in such eyes the real exciting cause is a fibrinoplastic inflammation.

Varieties and Manifestations of Sympathetic Inflammation.—Sympathetic ophthalmia occurs in several forms, sometimes arising in the wake of an attack of irritation, but frequently without any premonition or association of this character. It has been stated that marked oscillation of the iris often occurs when irritative phenomena are about to give place to an inflammation. Disturbance of vision is an early, if not the earliest subjective symptom of sympathetic ophthalmia, coming on in advance of the objective signs, but soon is followed by pericorneal injection, spots on Descemet's membrane, etc.

According to a classification adopted by many systematic writers sympathetic ophthalmia or, as it may be called, *sympathetic* or *infective uveitis*, because the uveal tract is especially involved, presents itself:

1. As an *iridocyclitis* (*uveitis fibrinosa sympathetica*, Schirmer), plastic or malignant—*i. e.*, an inflammation characterized by pain, photophobia, pericorneal congestion, discoloration of the iris, closure of the pupil by exudation around its margin and behind the iris, which is plastered to the capsule of the lens, cyclitis, precipitates on the posterior layer of the cornea, narrowing of the anterior chamber, rise of tension, effusion into the vitreous, involvement of the choroid, opacity of the lens, detachment of the retina, and finally shrinking of the eyeball.

2. As a *serous cyclitis* (Panas), or *serous iridochoroiditis* (de Wecker), or *serous sympathetic uveitis* (Schirmer), causing turbidity of the aqueous, deepening of the anterior chamber, punctate opacities on the posterior layer of the cornea, slight rise in tension, moderate ciliary injection, opacity in the anterior layers of the vitreous, and some involvement of the ciliary body and choroid. Frequently, if not in all the cases, *papilloretinitis* coexists with the uveitis. This process under proper treatment may cease and recovery result; but often it may be the forerunner of a malignant uveitis, with all its evil consequences.

Papilloretinitis (*sympathetic papilloretinitis*, Schirmer) as a coexisting condition in sympathetic ophthalmia has been noted. How frequent this association is it would be practically impossible to state because of the difficulty of making ophthalmoscopic examination in many cases. Rarely, according to Schirmer, it constitutes the primary affection. This papillitis usually is of moderate grade, the disk is not prominent, its borders are veiled and surrounded by grayish retinal opacity; the veins are swollen and tortuous, and occasionally small hemorrhages are present. Occasionally the swollen disk in very prominent, in one case studied by the author the appearances were those of high-grade choked disk.

A *choroiditis* caused by sympathetic inflammation originally recorded by von Graefe has been described a number of times, notably by Hirschberg, Caspar, Haab, and A. Dalén. The lesions somewhat resemble the spots of disseminated choroiditis due to syphilis, and appear, especially in the periphery of the eye-ground, in the form of small yellowish-red areas, with central pigment dots. According to

Dalén, the disease is a chorioretinitis, and not a pure choroiditis. A peculiar distribution of small yellowish-white flecks or nodes, lying behind the retinal vessels, often grouped and without pigmentation, has been described; they probably lie within the choroid. Choroiditis in the primarily affected eye has been recorded (Hirschberg, Heerfordt).

Other manifestations of sympathetic ophthalmitis have been reported, for example, a *simple atrophy of the optic nerve*, but the exact relationship of such a condition to a sympathetic affection has not been proved. In this connection reference should be made to the so-called *sympathetic amblyopia* described by Nuel, which, according to this author, begins at a much later period than true sympathetic ophthalmia—that is, at a period later than one or two months after the traumatic iridocyclitis has occurred. At first there is only a vague amblyopia, with obscurations; later visual acuteness is much reduced and the field of vision contracted, and should the amblyopia attain a decided degree, there may be a slight neuritis or pallor of the papillomacular bundle, or a perivasculitis. The affection may continue for months or even years, with alternate improvements and aggravations. Nuel explains it by assuming the presence of a neuritis caused by a hyperplasia of the interstitial tissues.

Premonitory Symptoms.—These are of great importance. One frequently described is tenderness in the ciliary region, frequently in a circumscribed spot, which may be detected by pressure with the end of a probe. The patient shrinks from the touch of the probe in a peculiar and striking manner. Sometimes an exactly similar tender spot is found in the ciliary region of the exciting eye. Biehler and E. von Hippel have demonstrated that fluorescein will color the endothelium of the cornea in certain uveitic inflammations when the superficial layers of the cornea are still intact and when ordinary examination fails to reveal these early changes. Alberti suggests the use of this test in cases of suspected but not yet manifest sympathetic inflammation. A. Maitland Ramsay and A. W. M. Sutherland have observed, using Bjerrum's method (see page 85), spindle-shaped enlargement of the blind-spot as a sign of active congestion of the optic disk, and suggest this examination as an important aid in determining the onset of trouble which may pass from an eye with an infected injury to the fellow eye. E. von Hippel has applied Abderhalden's test in cases of sympathetic ophthalmia; in these patients a positive biologic reaction to uveal tissue was obtained, but the reaction is not specific for sympathetic ophthalmitis. *Slit-lamp investigation* of the endangered eye may discover very early changes—punctate precipitation on the posterior surface of the cornea; also white "puncta" in the vitreous (A. Vogt).

The *general symptoms* of this condition are of importance. The presence of meningitis has been suspected, but never demonstrated. Severe headache, however, rise of temperature, delirium, and deafness have been reported, and it is of the utmost importance to submit patients who are suffering from sympathetic ophthalmitis to the fullest investigation from the general standpoint. H. S. Gradle's investiga-

tions indicate that marked *lymphocytosis* is likely to be present if an injured eye is of such character that it may produce sympathetic ophthalmia. The diagnostic and prognostic value of the test has been disputed by a number of observers. S. Gifford, for example, doubts if "mononucleosis" is specific for sympathetic ophthalmia; it represents, according to him, a reaction of the body to an inflammatory process in the eye. Gradle's own estimate is that while it would not be safe to rely upon the blood-count in deciding whether or not to enucleate an eye, the presence or absence of mononucleosis should be taken into account in doubtful cases. The blood should be examined for micro-organisms.

Period of Incubation.—The prodromal symptoms, in so far as the sympathizing eye is concerned, have been described. Although the exciting eye usually presents marked iridocyclitis, there are no phenomena which may be designated as pathognomonic. Based on the experience of the past war, and this applies also to the ocular accidents in civilian life, in general terms sympathetic ophthalmia is likely to occur if a plastic uveitis (iridocyclitis) arises as the result of a penetrating wound of the cornea or sclera, especially in the "danger zone," associated with prolapse of uveal tissue or lens-capsule. Persistent cyclitis or uveitis with gradual and increasing lowering of intra-ocular tension and of visual acuteness associated with photophobia of the fellow eye are signals of grave danger demanding enucleation of the originally injured eye.

The minimum time which elapses between the date of the injury and the development of sympathetic ophthalmia, save only in rare instances, is fourteen days; it must be feared during the first twelve weeks after the injury, especially between the sixth and the twelfth weeks; it is uncommon after this period, that is, after the third month. Exceptions to this general statement are: its reported incidence as early as the fourth day (de Lapersonne and Cahillous) and its postponement as late as twenty years and even longer.

Frequency.—The proportion of sympathetic ophthalmia to all ocular affections, that is, its relative frequency, has been variously stated (0.134 per cent. by Mooren, 0.15 per cent. by Becker). So also the proportion of sympathetic ophthalmia to severe penetrating ocular wounds has been variously estimated and is probably between 2 and 3.5 per cent., but it is not possible to be exact in this respect. Among those whose occupations do not expose them to eye accidents, children are more frequently affected than adults. Sympathetic ophthalmia, in spite of the numerous penetrating ocular injuries which the past war furnished, was uncommon as compared with its reported frequency during our own Civil War and during the Franco-Prussian War of 1870.

Four factors were potent in reducing to a gratifying minimum the incidence of sympathetic ophthalmia: (1) Accurate recognition of eyes so injured and inflamed that they should be sentenced to prompt excision; (2) proper early treatment of injured eyes; (3) the healthy general

condition of most of the soldiers; (4) abstinence from unnecessary minor operative procedures.

Pathologic Anatomy.—The pathologic anatomy of eyes which produce sympathetic ophthalmitis, that is to say, exciting eyes, has been thoroughly investigated, while that of the eyes which have become inflamed as the result of an infective cyclitis of the fellow eye, that is, sympathizing eyes, has not received much attention, for the simple reason that the opportunities of examination are rare. In so far, however, as they have gone, in general terms it may be stated that the lesions are identical; in other words, the same conditions are present in the exciting and the sympathizing eye.

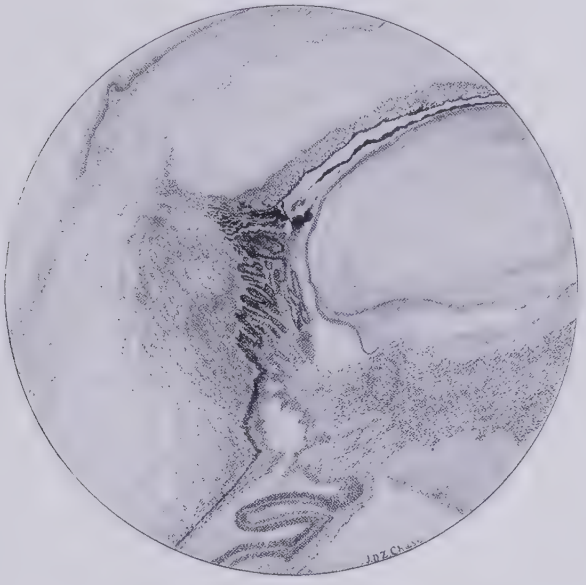


FIG. 168.—Traumatic iridocyclitis. Diffuse infiltration of the iris and ciliary body.

Schirmer has summarized the lesions in uveitis which produce sympathetic ophthalmitis somewhat as follows: All three portions of the uvea contain disseminated foci of small round cells, associated with a high grade of inflammation, characterized by a diffuse infiltration of the entire tissue with similar small cells. After the disappearance of the inflammation the uveal structure is destroyed and atrophied, and substituted by a pigmented connective tissue poor in vessels. Upon the surface of the iris and ciliary body there is a rich fibrinous exudation, with a strong tendency to organization, but the choroid fails to exhibit a similar exudative process.

Fuchs' investigations have thrown a new light on the pathologic anatomy of sympathetic ophthalmitis. He found in the uvea of exciting eyes a dense infiltration of lymphocytes, and in many cases in the midst

of this infiltration collections of epithelioid cells, often with giant-cells between them. This infiltration may be present only in certain spots in the form of isolated nodules in the iris, ciliary body, or choroid, or the uvea may be wholly or at least largely occupied by the process. Indeed, the infiltration may make its way into the sclera, which becomes permeated with scattered nodules. In so far as the different tissues of the eye are concerned and their implication in this process, the iris is the least affected, the ciliary body is always involved, and usually the choroid is more infiltrated than other portions of the uvea, especially in its posterior portion. A fibrinoplastic uveitis not infrequently complicates this *proliferative uveitis* of sympathetic inflammation, but Fuchs does not regard this as an essential factor in the process, because it may be absent in typical cases. Bacteria were not demonstrated in the nodules, but Fuchs does not doubt that the affection is produced by bacteria, which, instead of causing an acute suppuration, originate a chronic proliferation, and this inflammation has the property of being transmitted to the second eye through the circulation.

Fuchs' observations have been confirmed by Lenz, Kitamura, E. V. L. Brown, and a number of other observers. Ruge, however, denies that the anatomic conditions found in an eye exciting sympathetic ophthalmitis are characteristic, and while he does not doubt that there is a mixed infection in many cases, he holds to Schirmer's view that there is a pure sympathetic plastic exudation. Meller concludes that such marked specific changes cannot always be found in the exciting eye as would justify a diagnosis, but such insufficiency of histologic findings does not alter the view that there is in the first eye a specific morbid process similar to that by which the ophthalmia of the second eye is produced and which is distinctly different from post-traumatic inflammations. Now, while it may be too much to say that proliferative uveitis is pathognomonic of sympathetic ophthalmitis, there seems no doubt, to use the language of E. V. L. Brown, that it is the essential anatomic condition present in an eye which causes sympathetic inflammation of its fellow.

Pathogenesis of Sympathetic Ophthalmitis.—Formerly it was almost universally thought that this disease was due to a reflex action transmitted through the ciliary nerves, and on this theory the name "sympathetic" was adopted. The exact nature of this grave malady is not known, although the older hypotheses have largely been abandoned for the *theory of infection.*

According to Deutschmann, the inflammation is a progressive process in the continuity of the tissue of one eye to the other by way of the optic nerve apparatus, and is of bacterial origin; hence, a *migratory ophthalmitis.* Deutschmann's researches have, however, not been confirmed (Gifford, Mazza, Randolph, Limbourg, Levy, and Greeff). Römer's investigations have utterly set aside the possibility of accepting the ciliary nerve theory in any form. Bellarminoff and Selenkowsky believe that all cases of sympathetic disease may be explained

by the action of a toxin which is produced by the bacteria which have entered the primarily affected eye, and which reach the other eye by means of the lymph and diffusion streams. Brown Pusey suggests as an explanation of sympathetic ophthalmitis that the cells of the injured eye, probably those of the ciliary body and iris, give rise to a cytotoxin, which, having a selective affinity for corresponding cells in the other eye, there sets up an inflammation. A precisely similar theory has been propounded by Golovine. Motais, although frankly stating that he has no clinical evidence to support him, and that as yet no experimental researches are at hand to lend aid to his hypothesis, maintains that the anastomoses between the veins of exit of the eyes constitute the most probable paths for the transmission of sympathetic ophthalmitis; and even so great an authority as Leber believed that this view should receive consideration. Römer regards sympathetic ophthalmitis as a *metastasis*, the metastatic infection proceeding by way of the blood-streams, and thus brings himself in accord with views originally expressed by Berlin. This infection is ascribed to some form of micro-organism which is pathogenic for the eye alone and does not affect the body generally. The metastasis theory, according to Römer, most satisfactorily explains all of the phenomena of so-called sympathetic ophthalmitis, and is entirely consistent with the result of modern bacteriologic research. Moreover, it has now received confirmation from the anatomic standpoint, especially by Fuchs' researches.

The micro-organisms which excite sympathetic ophthalmitis have not been identified; indeed, they have not been seen, and, therefore, it may be assumed that they are distributed in invisible form by the intra-ocular fluids throughout the eye, to which they have been transmitted by the blood-streams.

While the prevailing theory is that in sympathetic ophthalmia the infection enters the eye through a wound or other pathway, J. Meller has advanced an *endogenous theory*. According to him some part of the body other than the eye is the port of entry for the specific organism which has an elective affinity for uveal tissue, and attacks that of the diseased eye because its resistance is lowered, the damage having occurred from a wound or a toxic iridocyclitis. Gradually sympathetic inflammation arises, which leads to metastasis to the other eye. E. V. L. Brown believes that this theory explains those cases in which in the primary eye neither wound nor other opening can be found.

Recently Elschnig has maintained that in the development of sympathetic ophthalmia two factors are necessary: *anaphylaxis* of uvea, on account of tissue disintegration, the chief rôle being taken by the pigment, and an anomalous condition of the organism, for example, nephritis, diabetes, auto-intoxication, etc. Naturally, this theory of the existence of *anaphylactic uveitis* has met with much opposition, but has acquired some advocates, for instance, A. Jess, who admits it explains certain clinical facts better than the bacterial hypothesis, to wit, the period of incubation, and Cramer, who observed a second attack of sympathetic ophthalmia coincident with *alopecia* and *whitening of*

the eyebrows. This he believed was an anaphylactic reaction of pigment origin.

The greater portion of Elschnig's experimental work has been confirmed by Alan C. Woods. He has further shown that if uveal pigment is absorbed from the eye, following an injury to the uveal tract, either pigment antibodies may appear in the blood-stream (the formation of an apparent immunity to uveal pigment) or else the body may become definitely hypersensitive to the uveal pigment. The development of the circulating pigment antibodies is usually accompanied by a more or less normal healing of the injured eye, whereas the development of the tissue hypersensitivity to pigment appears to be closely associated with the development of sympathetic ophthalmia in the second eye. The presence or absence of the antibodies in the blood-stream may be detected by a complement-fixation reaction on the blood-serum. When these antibodies are found, and the prognosis may therefore be assumed to be good, Woods calls the result "positive." When the antibodies cannot be demonstrated, the reaction is called "negative." Woods has, however, emphasized the fact that this reaction is very delicate, and does not believe that in its present form it is susceptible of wide clinical application. The presence or absence of a tissue hypersensitivity may be detected by an intradermal skin test with uveal pigment.

Treatment.—The most important consideration is *prophylaxis*, or, in other words, the management of the eye originally affected. This depends upon the character and situation of the wound or upon the stage of the disease, and upon the amount of vision possessed by the injured or diseased organ.

In the section devoted to Treatment of Wounds of the Sclera (see page 326) the method is described by which eyes seriously wounded may be saved. Schirmer believes that the treatment of injured eyes should include full doses of mercury, and the author can confirm the therapeutic value of this remedy in this respect, as well as the value of large doses of salicylate of sodium.

Where every advantage of nursing and careful watching is at hand, eyes may be saved which would be sacrificed in other circumstances. The propriety of operating may be determined by regarding the following rules, which represent the published experiences of the best authorities.

Enucleation should be performed on—

1. An eye with a wound so situated as to involve the ciliary region, and so extensive as to destroy sight immediately, or to make its ultimate destruction by inflammation of the iris and ciliary body reasonably certain.

2. An eye with a wound in this region already complicated with severe inflammation of the iris or ciliary body, even if sight is not destroyed; or an eye containing a foreign body which judicious efforts have failed to extract, and in which severe iridocyclitis is present, even if sight is not destroyed.

3. An eye the vision of which has been destroyed by plastic iridocyclitis, or one which has atrophied or shrunken, provided there are tenderness on pressure in the ciliary region and attacks of recurring irritation; or without waiting for signs of irritation.

4. An eye the sight of which has been destroyed, even though inflammation has begun in the sympathizing eye, because by this means a source of additional infection is removed, and the treatment of the second eye is rendered more effectual.

5. An eye in which the wound has severely involved the cornea, iris, or ciliary region, the fellow eye being in a state of persisting irritability or subject to frequent attacks of so-called sympathetic irritation.

6. An eye either primarily lost by injury or in a state of atrophy, associated with signs of so-called sympathetic irritation in the fellow eye (see also page 370).

It is universally conceded that the enucleation of an eye (*preventive enucleation*) primarily injured, the visual function of which cannot be restored, is the surest way of preventing a sympathetic ophthalmitis. It must be remembered, however, that even a very early enucleation does not necessarily prevent sympathy in the fellow eye, because the infective process may have begun before the operation, and may not develop for several weeks. It has occurred fifty-three days after the enucleation of an eye injured twenty days prior to its removal (Stephenson), and C. B. Welton has collected 28 cases of this character, the shortest interval between the injury and the enucleation being nine days and the longest two months. In place of enucleation, evisceration has been practised, but this operation has been followed by sympathetic inflammation. Resection of the optic nerve (neurectomy) does not provide security; enucleation is the only satisfactory procedure.

If sympathetic inflammation has begun, the rules recorded are subject to modification in that *enucleation must not be performed if there is any vision in the exciting eye*, which in the end may prove to be the more useful organ. The treatment already recommended for iritis and iridocyclitis is appropriate.

Iridectomy has been advised when the inflamed iris is completely bound to the lens-capsule, but it is better to await the subsidence of acute symptoms before attempting any surgical interference unless the intra-ocular tension is inordinately raised, and of itself is a factor which must be dealt with. Scleral incision has been practised in these circumstances, but probably an iridectomy would be the better procedure.

The *general treatment* consists in confinement in a darkened room (moderate exercise with eyes well protected is permissible in subjects failing for lack of it); complete functional rest of the eyes and atropin or scopolamin locally, provided there is no rise of tension and no atropin irritation; and leeches to the temple if the inflammation is florid. Dionin (5 per cent.) should be used. Mercurial inunctions are important,

and free diaphoresis, either by the administration of pilocarpin or by vapor baths, has been advised; in debilitated patients tonics and alteratives are useful. The value of sodium salicylate, advocated by Gifford in the treatment of sympathetic ophthalmitis, is great, at least 60 to 100 grains (3.9–6.5 gm.) a day should be exhibited, and even larger doses are sometimes well borne; but, inasmuch as the treatment must continue for long periods of time, the doses must be regulated strictly according to the results achieved. Gifford believes that most patients are able to take daily 1 grain (0.065 gm.) of sodium salicylate for each pound of weight; but in rebellious cases advocates massive doses, even as much as 200 to 300 grains *per diem*. In place of salicylate of sodium, acetyl-salicylic acid (aspirin) and benzosalin may be used. While sodium salicylate cannot prevent sympathetic uveitis, its use during traumatic iridocyclitis seems to render the sympathetic affection less virulent than otherwise would be the case, and in this respect is the superior of mercury. Gifford has found atoxyl to be useful, and recently has advised large doses of atophan, 40 to 60 grains (2.6–3.9 gm.) a day especially when the salicylates are not well born. The administration of salvarsan and neosalvarsan or their equivalents have been tried and favorable results have been reported; indeed, the good results in some of the cases furnished by the past war were attributed to these remedies (Morax). The author has observed encouraging improvement from arsphenamin treatment; massive doses of salicylate of sodium and free diaphoresis were also employed. The success of salvarsan therapy suggests to some observers that sympathetic ophthalmia may be *protozoal* in origin. Bernheimer reports good results in one case from the injection of tuberculin. Because the uveal infiltration in sympathetic ophthalmia resembles that of tuberculosis, the possibility of a dormant tuberculosis as a factor in producing this disease has been emphasized by H. H. Stark. Intra-ocular injections of bichlorid of mercury, in the opinion of the author, should not be employed, although they have been highly endorsed. Subconjunctival injections have been recommended. The author's experience with them in this disease has not been encouraging. G. S. Derby and H. N. Pratt conclude, from their own experience and that of Zur Nedden, that the blood-serum of an individual with sympathetic ophthalmia may be of curative value when injected into another patient with the same disease. Not less than 1 ounce (30 c.c.) of the serum was used. Naturally, the possible influence of focal infections on sympathetic ophthalmia has not escaped attention (Harbridge), and E. V. L. Brown has reported improvement in the disease after removal of infected tonsils. A. Knapp, discussing the autotoxic factor in sympathetic ophthalmia, has recorded cases not yielding to ordinary remedies, but helped when a marked intestinal sepsis was eliminated. Areas of focal infection should be eliminated. As deficient thyroid secretion is believed to lower the resistance of tissue to infective processes (Dunn) thyroid extract has been suggested —a remedy which the author tried in one case with advantageous effects, which, however, were only temporary. Woods, in collaboration

with Arnold Knapp, has taken advantage of the findings previously described (page 378), namely, that the development of an immunity to pigment protects against a sympathetic ophthalmia, while the development of a tissue hypersensitivity to pigment is associated with the disease, and treated cases of sympathetic ophthalmia by injections of uveal pigment. This form of treatment consists in desensitization with the pigment, and the subsequent continuance of the injections until a state of immunity is produced. This treatment is too new as yet to allow the expression of any opinion as to its merit, but the published reports of cases so managed indicate that it may be of definite value in conditions which otherwise appear hopeless.

Van Lint and Coppez have adopted the method known as the *formation of a fixation abscess* in the treatment of intractable iridochoroiditis and sympathetic ophthalmitis. One c.c. of pure oil of turpentine is injected in the subcutaneous tissue of the flank. Usually in a few days an abscess filled with aseptic pus develops. About the seventh day the abscess is incised and the contents evacuated. Although the authors state that the procedure does not produce a cure, it had in every instance led to an arrest of the progress of the malady.

As the result of treatment the affected eye may recover with useful sight, or pass into atrophy or phthisis bulbi, or grow quiet, with the formation of complete annular adhesions of the iris to the capsule of the lens, which has become cataractous.

To improve vision under the last-named condition iridectomy and iridotomy have been tried, but the results are usually unfavorable. Extraction of the cataractous lens, with iridectomy, also presents serious difficulties. For those cases in which a transformation of the iris, lens and capsule into a tough, opaque, and inelastic tissue has occurred, Mr. George Critchett practised the following operation: A speculum is introduced, the globe is fixed, and a fine cutting needle is introduced through the cornea, its point being directed to the center of the capsule. This structure is penetrated by making a rapid rotary movement, on the principle of a gimlet. A second needle is introduced from the opposite side and the points separated from each other, the result being a rent in the center of the capsule and the escape of the soft lens matter. The operation must be repeated at proper intervals until a clear pupil has been obtained. It is suited to young eyes, although it may succeed in adults, as in one case in the author's practice. Care should be taken to avoid wounding the iris. With this operation the author has achieved gratifying success.

The **prognosis** of sympathetic ophthalmitis, while it is essentially grave, is certainly not as unfavorable as in former times, probably owing to modern antiseptic procedures, and to improved methods of treatment. H. Gifford believes that "75 per cent. of the cases, if seen within the first week, retain useful sight if properly treated." While complete and permanent recovery occurs, the patient cannot be considered cured until at least a year has elapsed.

Unless the disease is recognized at its very beginning and vigor-

ously and properly treated, the sight of the eye is lost and the organ shrinks. Excepting the cases of pure papillitis (Schirmer), those varieties which appear as a so-called serous uveitis, and which retain this character of inflammation, afford the most satisfactory prognosis. It is extremely important to warn patients of the grave nature of this malady, and if an attempt is made to save an eye injured in the manner already described, it must be done with the full understanding of the risks which are undertaken, and the patient must be kept under constant observation.

CHAPTER X

DISEASES OF THE CHOROID

Congenital Anomalies.—Two striking congenital anomalies occur in connection with the choroid:

1. *Coloboma of the choroid* is a large defect in the choroid, almost always in its lower part, and often associated with a similar vice of conformation in the iris. Other ocular abnormalities which may be found with coloboma of the choroid are persistent pupillary membrane, curvature defects of the cornea, strabismus, nystagmus, microcornea, and persistent hyaloid artery. Coloboma of the lids, harelip, and dermoids may be coincident anomalies. The influence of heredity has been emphasized in relation to coloboma of the choroid and iris (Arlt, de Beck, C. E. G. Shannon).

Examined by means of the ophthalmoscope the deficient area appears as a glistening, pearl-colored patch, often irregular on its surface, owing to the development of several protrusions and corresponding intervening depressions, and bordered by an irregular pigment line. In some cases the retina may be recognized as a translucent veil covering the defect, and the retinal vessels occasionally pass into the depression; in others the retina is absent, and the defect is represented in the visual field by a scotoma. The coloboma may include the optic nerve entrance, either partially or completely, or may be separated from it by a bridge of healthy choroid. It may be confined to the area around the disk, or pass downward as far as it can be followed, and may be associated with a similar defect in the iris, from which it is separated by a band of choroid tissue. Sometimes several defects are present in the same eye-ground. Imperfect closure of the *fetal cleft* (*choroidal fissure*) is the usual explanation of this condition. Some choroidal defects resembling coloboma apparently depend upon an intra-uterine choroiditis. According to E. T. Collins and W. Lang, all colobomas of the choroid may be explained by assuming that they are due to an abnormal adhesion of the retina to the mesoblast, which may take place either before or after the closure of the fetal cleft.

In addition to coloboma in the usual situations, similar defects have been described in the macular region (*macular coloboma*, see Fig. 178) and in the nasal half of the eye-ground (B. A. Randall and the author), and for these defects, which do not involve the optic disk, Lindsay Johnson has proposed the name *extrapapillary coloboma*. *Atypical coloboma of the choroid* may also be situated upward and outward and probably in any part of the choroid. It is due to failure in the formation of blood-vessels in the inner part of the mesoblast surrounding the secondary optic vessel (Collins and Mayou).

A striking congenital abnormality is one in which the entire choroid and pigmented epithelium except over a small area in the region of the macula is absent. The red reflex of the fundus being absent an extensive white stretch is visible. To this condition the name *choroideremia* has been given. Both eyes are affected; the patients are night-blind (Nettleship). The direct vision may be normal, but also defective partly as the result of high refractive error, as is the case in one patient observed by the author. The condition has been well studied in this country by A. B. Connor.

2. *Albinism*, or a congenital want of pigment in the choroid and iris, is a deformity met with both in a *complete* and *incomplete* form. It depends upon a failure of pigment-development in mesoblastic tissue sur-

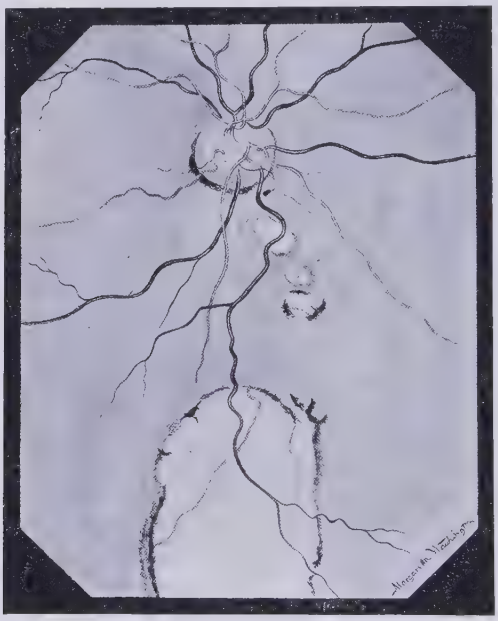


Fig. 169.—Congenital defects in the choroid; one large coloboma in the usual situation, with two smaller areas between it and the disk.

rounding the secondary optic vesicle and in the outer layers of the secondary optic vessel (Collins and Mayou).

The iris has a pink or pink and yellow appearance, due to the reflection of light from its own blood-vessels and from those of the choroid, which, in the most pronounced forms of the defect, can be seen with the ophthalmoscope down to their finer branches. The anomaly is most marked in early childhood, is almost invariably associated with lack of pigmentation in the hair, and is accompanied by nystagmus, amblyopia, and high grades of refractive defects. Usually these eyes are photophobic. In many instances albinism has been observed in several members of the same family, and seems to be hereditary. *Semi-albin-*

ism, especially in the periphery of the fundus, is sometimes observed in infants and may persist in adult life.

Hyperemia of the Choroid.—An actual hyperemia could be demonstrated only by finding a real distention of the vessels of the choroid, which usually are invisible, and the *congestion of the choroid*, described as being present in myopic or asthenopic eyes, and as the result of exposure to bright light and heat, is more often a figure of speech than a proved pathologic condition.

In eyes subjected to prolonged strain, the result of uncorrected ametropia, certain changes in the normal appearance of the fundus arise which are usually described under the vague term "choroidal disturbance." We may, perhaps, assume hyperemia where the nerve-head presents distinct redness, which is imperfectly differentiated from the unduly flannel-red appearance of the surrounding choroid, or where the choroid, instead of exhibiting its usual uniform red color, has changed into what has been denominated a "woolly choroid," with faint dark areas in the periphery, indicating the interspaces between the choroidal vessels, and more or less pronounced retinal striation surrounds the disk. This is a familiar picture in many cases of "eye-strain." Sometimes the ophthalmoscope reveals, in addition to the other signs already described, an appearance as if fine pigmented grains had been scattered over the fundus, especially its central regions. To this manifestation Theobald has given the name "miliary choroidoretinitis." Similar lesions may follow exposure to great heat and light, and may be seen in the eyes of puddlers, glass-blowers, etc.

Treatment.—In this condition, often associated with the subjective symptoms of aching eyes, some intolerance of artificial light and distinct asthenopia, the eye should be atropinized, dark glasses should be worn, and after the irritable condition of the fundus has sufficiently subsided a proper correction of the refractive error should be ordered. Internally, small doses of iodid and of bromid of potassium serve a useful purpose.

Choroiditis.—Under the general term *choroiditis* are included various types of inflammation of the choroid.

Causes.—Choroiditis, like iritis, may depend upon constitutional disorders, infections, toxins, and traumatisms, or upon disease in other portions of the eye. Choroiditis is often classified according to the probable etiology—for example, *syphilitic*, *tuberculous*, *traumatic*, etc., choroiditis. (Compare with pages 326, 327.)

Symptoms.—Certain symptoms, for the most part revealed only by the ophthalmoscope, are present:

1. Alteration in the uniform dull red surface of the eye-ground caused by (*a*) the absorption of the pigmented epithelium; (*b*) patches of pale yellow or grayish-white color with ill-defined boundaries due to exudation (*recent choroiditis*); (*c*) patches of white color due to exposure of the underlying sclera (*atrophic choroiditis*); and (*d*) patches of black pigment, variously shaped, scattered over the fundus, and usually bounding the spots of atrophy (*pigment heaping*).

2. Absence of external manifestations indicative of the deep-seated disease, except where acute and purulent forms are accompanied by injection, chemosis of the conjunctiva, etc.

3. Changes in the transparent media (lens, vitreous) by the formation of opacities, as a secondary result of the choroidal disease.

Subjective symptoms peculiar to choroiditis do not exist.

Pain usually is not present except in purulent forms, and in such varieties as may be complicated with iritis, or as the result of associated refractive error.

Disturbance of vision is in direct relation to the situation of the lesions and the amount of atrophy. If the choroidal disease is peripheral, visual acuteness may be unaffected; if atrophic patches occupy the macular region, central sight may be gently diminished or practically obliterated. It is remarkable, however, that even in extensive diffuse choroiditis good vision may be retained. If the disease has caused secondary changes in the vitreous or lens, these add to the depreciation of visual acuteness.

Scotomas, both positive and negative, may be present. The field of vision is contracted in many types of choroiditis, especially if secondary atrophy of the optic nerve has occurred. The displacement of the retinal elements overlying the diseased choroidal areas causes *metamorphopsia;* sometimes objects appear smaller than they really are, *micropsia;* sometimes larger, *macropsia.* In the early stages of choroiditis the patients are often much annoyed by subjective symptoms of light—*i. e., photopsies.*

Diagnosis.—This is readily made by observing with the ophthalmoscope the appearances briefly summarized in paragraph 1 of the general symptoms.

Inasmuch as choroiditis, in the large majority of cases, is associated with retinitis, it is difficult to decide whether the pigment lies in the choroid or retina. If the pigment mass is covered by a retinal vessel, and at the same time is situated in a deeper layer than this, its position is judged to be in the choroid; if the retinal vessel is covered by the pigment mass, and the latter is situated more anteriorly, its position is assumed to be in the inner surface of the retina, to which spot it has wandered through secondary involvement of the retina. Pigment characterized by a "lace-like pattern," or resembling bone-corpuscles, is always in the retina (Nettleship). Naturally, it is not uncommon to find pigment spots and masses occupying both a retinal and a choroidal position in the same eye-ground.

Course, Complications, and Prognosis.—A choroiditis may be sudden in onset and pursue an acute course; for example, an acute choroiditis at the posterior pole of the eye resulting in a permanent myopia (see also page 139), or purulent forms of the disease.

More commonly the course of choroiditis is slow and chronic. Beginning with exudation or hemorrhage, it passes by gradual stages through the period of absorption, atrophy, and pigment accumulation.

It is by the last signs that a former choroiditis is recognized, and the changes are called "old choroiditis" or "choroidoretinitis."

The following structures are liable to become involved during the course of a choroiditis: The retina, which from its intimate association with the choroid through the pigmented epithelium probably does not escape in any instance, and in many the association of disease is so close that we apply the term *choroidoretinitis* or *retinochoroiditis;* the optic nerve (*choroiditic atrophy*); the vitreous (*vitreous opacities*); the crystalline lens (*posterior cortical and polar cataract*); the iris (*iridochoroiditis*); and the sclera (*scleroticochoroiditis*).

The *prognosis* in choroiditis is always grave, and although careful treatment may preserve sight, in many instances great depreciation of vision and even blindness may ensue. Necessarily the prognosis as to vision depends upon the position of the disease and its relation to the macula.

Pathologic Anatomy.—In non-purulent forms of choroiditis collections of round cells are gathered in the choroid, especially along the vessels between this membrane and the retina, and hemorrhagic extravasations may be seen. Organization of this round-cell exudation causes atrophy of corresponding portions of both choroid and retina, union of the two membranes, disappearance of the pigment layer of the retina except at the edges of the lesions, where it is proliferated, and wandering of the pigment cells into the retina along the lines of the vessels. In purulent choroiditis there are a dense cellular infiltration of the choroid, rapid involvement of retina and vitreous, panophthalmitis, and subsequent phthisis bulbi.

As already stated, choroiditis may be *acute* or *chronic*, and at one time was classified, according to the pathologic conditions, into *plastic*, *serous*, and *purulent* forms.

For the present purpose choroiditis may be divided into *superficial* and *deep* choroiditis, and a well-recognized classification may be adopted which places all forms under one of two heads: (1) *Non-suppurative exudative choroiditis* and (2) *suppurative choroiditis* and *iridochoroiditis*.

Treatment.—This, in general terms, demands perfect rest for the affected eye, protection from glaring light, and the administration of remedies indicated by the cause of the choroiditis. Further details are reserved for the sections devoted to the several varieties of choroiditis.

Superficial Choroiditis (*Epithelial Choroiditis*).—Instead of the general dull red appearance of the eye-ground, the larger vessels may be manifest as rather broad, reddish, or yellowish-red stripes, which traverse the fundus in an interlacing manner, and between which are dark intervascular spaces, many of them having a lozenge-shaped appearance. This is due to the absorption of the pigment epithelium and the capillary layer which lies just beneath it.

In certain instances these appearances are physiologic, and are commonly visible in the periphery of eye-grounds, often by preference occupying a space down and in from the disk.

They may be universal, the only portion of the eye-ground escaping being the region directly confined to the macula, and in such circumstance a striking ophthalmoscopic picture is evident. The larger vessels of the choroid-stroma pass in a sinuous manner across the eye-ground, bringing out into distinct relief the pigmented connective-tissue cells of the choroid proper which lie between them (consult Fig. 178, page 398). The atrophy is superficial and of itself does not disturb vision. Such lesions are seen in myopic eyes; in "stretching eyes," where hyperopic refraction is diminishing or passing into myopic refraction; in glaucoma; and sometimes are associated with retinal disease—for example, pigmentary degeneration.

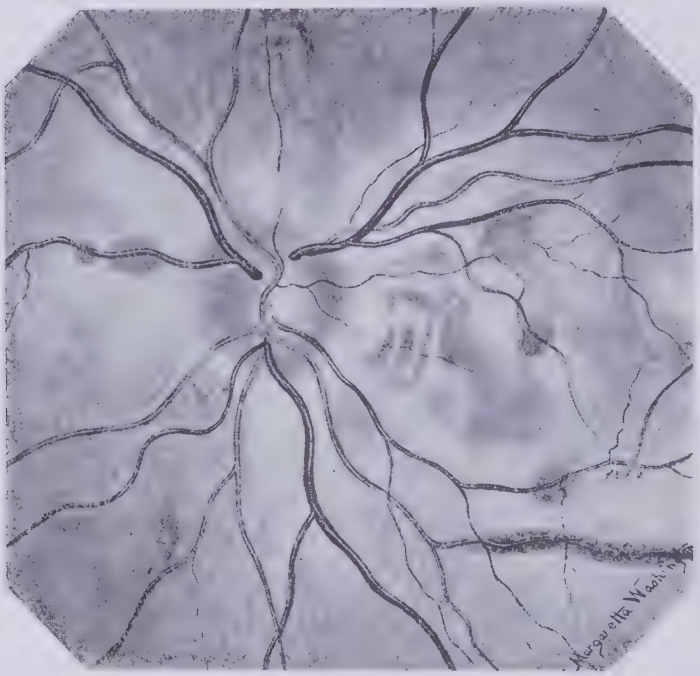


FIG. 170.—Acute choroiditis with wide-spread, fog-like exudations which subsequently gave place to areas of atrophy and pigment heaping.

Deep Choroiditis.—1. **Diffuse Exudative Choroiditis.**—This occurs in an *acute* and in a *chronic* form—*i. e.*, the chronic form represents the ophthalmoscopic appearances commonly observed after subsidence of the acute process.

In the early stages of *acute* or *recent choroiditis* the diseased areas are represented by yellowish-white, sometimes greenish-gray, exudations, which may be diffuse, circumscribed, or disseminated, and which shade gradually into the surrounding eye-ground, or which may be fringed with pigment, and by small eroded areas, and hemorrhages.

Later these areas of exudation undergo absorption and metamorphosis, and some of the following conditions are present, which may be

named *chronic choroiditis*. Instead of the normal red of the eye-ground the ophthalmoscope reveals white or yellowish-white plaques, sometimes separated by partly normal choroid, more often running into one another until a huge expanse of exposed sclera is visible throughout the fundus.

The white patches appear speckled because numerous pigment masses of black color are collected upon them, irregular in form, sometimes gathered in lumps, sometimes assuming variously shaped groups. They lie beneath the retinal vessels for the most part, although usually pigment will be found collected upon these retinal vessels showing the participation of the retina in the process (*choroidoretinitis*, Fig. 171). In other patches the atrophy has not been sufficient to expose the glistening white sclera, and there will be found the lesions of superficial choroiditis, namely, band-like, orange-yellow, or light red vessels, freely anastomosing with each other, and, between them, the pigmented epithelium. In still other spots yellowish exudations are evident, which represent the earlier stages of the process already described. In these circumstances all the stages from yellowish extravasation to complete atrophy are visible.

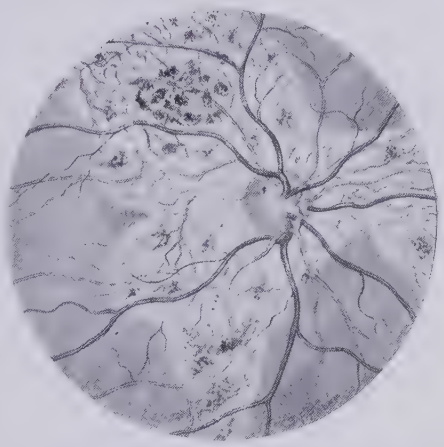


FIG. 171.—Diffuse exudative choroiditis with choroidoretinitis.

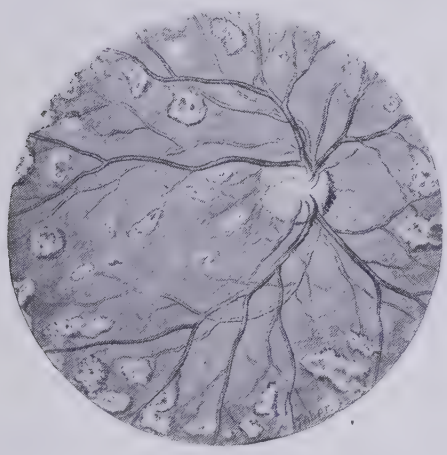


FIG. 172.—Disseminated choroiditis.

2. **Disseminated Choroiditis.**—Another form which may be looked upon, according to a classification adopted by some authors, as the circumscribed variety of the type just described, is that which is known as *disseminated choroiditis*.

In this type, usually beginning in the periphery, but gradually approaching the center of the eye-ground, numerous round or oval spots surrounded by black margins are found. The white center of the spot is the exposed sclera; the black margin, the altered pigment. Instead of a white center there may be a single black mass, in its turn

encircled by a yellowish ring. Where the spots assume a punched-out appearance, as if a sharp instrument had cut out the tissue down to the sclera, the margins of the incision being bordered with pigment, they are characteristic.

These spots of disseminated choroiditis vary greatly in number. There may be only one or two, or the eye-ground may be dotted over with them. Between the spots the choroidal tissue is comparatively healthy. The earlier stages of such spots consist in small, yellowish or greenish-gray exudations, which gradually absorb, leaving the atrophic marks which have just been described (Fig. 172).

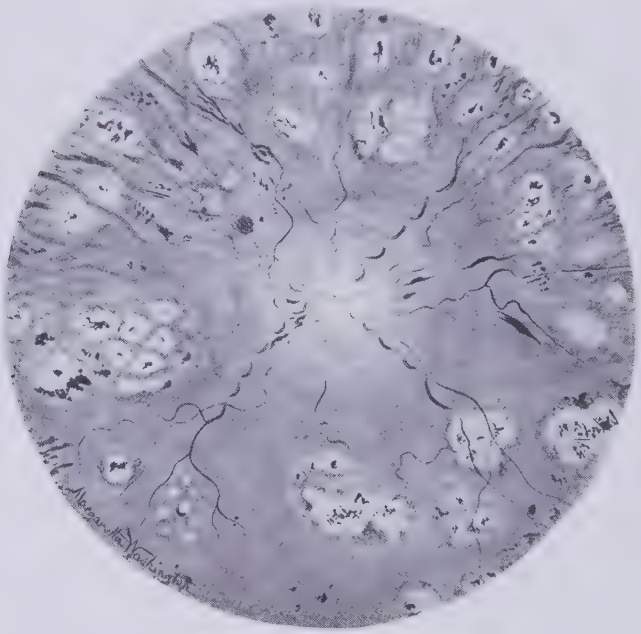


FIG. 173.—Disseminated choroiditis and optic neuritis with retinal hemorrhages in a syphilitic patient in the Orthopedic Hospital. The choroiditis is of long standing, the neuroretinal lesions a fresh implantation.

Vitreous opacities are often present, either faint and floating, or large, string-like and membranous. There may also be cataract at the posterior pole of the lens.

The optic nerve may be affected in the later stages of deep choroiditis and undergo a species of atrophy to which the name *choroiditic atrophy* has been applied. The edges of the disk are slightly hazy, the color a reddish yellow, and there is contraction of the retinal vessels. Disseminated and other forms of choroiditis are often associated with *secondary pigmentation of the retina*, and the pigment patches not uncommonly resemble those seen in pigmentary degeneration of the retina. These pigmentations are especially noteworthy in forms of disseminated choroiditis due to syphilis. One form of disseminated

choroiditis with neuritis and peripapillary retinal opacity may be associated with a focus of tuberculosis in the optic nerve (Gilbert, Michel).

3. **Circumscribed Plastic Choroiditis** (*Localized Exudative Choroiditis; Choroiditis with Descemetitis*).—Areas of choroiditis are not infrequently encountered in young persons—that is, from fifteen to thirty years of age—which ophthalmoscopically do not differ materially from those types already described, except in their circumscribed character. Usually there is a large, bluish- or grayish-white patch of effusion, generally denser in its center and thinning off at its margin into the healthy fundus. The diseased area may be close to the disk (Hill Griffith), near or at the macula, or in the periphery (Friedenwald), even in the far periphery. The early exudative stage is followed by erosion and atrophy, but the course is comparatively benign, and, unless the macular region is involved, there is no distinct depreciation of central vision. Not uncommonly keratitis punctata (descemetitis) and vitreous opacities accompany the condition (see also page 363). Not infrequently rise of intra-ocular tension may be present and an attack of *acute glaucoma* may be precipitated, especially if a mydriatic is employed. Relapses may occur in the sense of reappearance of an exudation closely adjacent to the patch of atrophy remaining after the subsidence of the original lesion; or the lesion may be located at some other part of the eye-ground. It is not an infrequent experience to find irregular patches of atrophy fringed or spotted by pigment which represent areas of former circumscribed choroiditis of this character. Plastic choroiditis near the disk may give rise to an appearance similar to that of optic neuritis.

Retinochoroiditis juxtapapillaris, first described by Jensen as a clinical entity, is characterized by a prominent infiltration close beside the nerve head, approximately the size of the papilla (sometimes larger) and usually oval in shape, which wholly or partially obscures the retinal vessels. The choroid is involved; opacities form in the vitreous. The defect is interpreted in the field of vision by a scotoma, sector-like, in that it extends from the blind-spot to the periphery.

Jensen describes the fundus oculi as otherwise normal, but pigmentary changes have been reported in the opposite eye (Groes-Petersen) and in the macula in the corresponding eye (Van der Hoeve). Precipitates on the back of the cornea have been reported. The disease slowly subsides and leaves an area of atrophy. Recurrences may take place. Jensen found no certain etiologic factor. The author noted persistent anemia in one case. Verhoeff, who examined an eyeball of a syphilitic patient and found a granulomatous lesion centered near the disk margin involving the inner layers of the retina and secondarily the choroid and disk, suggests that Jensen's retinochoroiditis may sometimes be specific in origin. This certainly is not the cause in the majority of cases. It has been attributed to tuberculosis and to focal infections. The disease occurs in young persons.

4. **Anterior Choroiditis.**—To this condition reference has been made in the description of parenchymatous keratitis (see page 299).

The lesions are situated far in the periphery, and may exist as a special form of disease, or result from an extension backward of affections of the iris and ciliary body.

Causes.—The cause of deep choroiditis, either diffuse or disseminated, is acquired syphilis in a number of cases, and the disease appears from six months to two years after the initial infection. Sometimes it is postponed to a much later period (tertiary period). Opacities in the vitreous are common in syphilitic choroiditis. Although certain choroidal lesions have been looked upon as especially characteristic of syphilis, it is not safe to attempt to make a diagnosis of syphilis simply by the appearances of any of the varieties of choroiditis (compare with syphilitic chorioretinitis, page 487). Diffuse syphilitic choroiditis depends upon the toxin of syphilis, or perhaps a dissemination of its active agent (Spirochæta pallida) throughout the tissue of the choroid. If the deposit of the toxin remains localized the circumscribed varieties of the affection arise. Disseminated choroiditis, choroidoretinitis, and secondary pigment degeneration of the retina are seen in children the subjects of hereditary syphilis. Choroiditis due to acquired syphilis usually affects both eyes. The Wassermann test should be applied to all cases of choroiditis. Igersheimer maintains that *typical* disseminated choroiditis (page 389) is less commonly caused by syphilis than is usually supposed to be the case. To vitreous opacities in *typical* disseminated choroiditis as an indication of its luetic origin, on which Alexander insists, Igersheimer is unwilling to attach importance. He, however, does not eliminate syphilis as a causative factor in many forms of this disease.

A disseminated choroiditis (*hereditary choroiditis*) affecting both eyes is occasionally encountered as a family disease independently of syphilis and associated with disorders of the central nervous system (Hutchinson), and *familial chorioretinitis* has been observed. Patches of choroiditis are found in the eyes of children born with cataract.

A choroiditis quite indistinguishable from the forms described may result from an injury (*traumatic choroiditis*). As a complication of concussion injuries of the globe *pigmented choroiditis* may arise in the form of scattered areas of variously shaped pigment masses, usually of small size, sometimes granular, interspersed with small erosions and spots of atrophy (see also page 409).

Many types of choroiditis, especially of the *disseminated* variety, as well as diffuse and localized exudative manifestations, are due to tuberculosis (*tuberculous choroiditis*). The influence of tuberculosis in the development of choroiditis is one which demands close investigation, and any patient whose eye-ground reveals on ophthalmoscopic examination choroidal lesions should be submitted to a thorough general examination. This etiologic factor can be usually demonstrated by tuberculin injections, which are followed by local as well as general reaction, but it must be remembered that focal reaction, while highly suggestive, does not prove that the ocular disease is tuberculosis.

Choroiditis has also been ascribed to disturbances of nutrition,

metabolic disorders, nephritis (*renal choroiditis*), diseases of the liver (*ophthalmia hepatica*), anemia, chlorosis, acute infectious diseases, and to infections arising from the nasopharynx, accessory sinuses, and the teeth (pyorrhea alveolaris). In acute plastic choroiditis the following etiologic factors are active: focal infections, for instance, tonsillitis, oral sepsis, intestinal toxemia, etc., auto-intoxication, typhoid fever and other infectious fevers, influenza, and pneumonia. Gradle suggests that the infecting material from the accessory sinus or other focus of infection may gain access to this region through the posterior ciliary vessels. (Additional causes, see page 361.)

The **prognosis** is always grave if the process is an extended one and the macula involved; it is best in the syphilitic cases and in some forms of acute plastic choroiditis, in which the results of treatment are most satisfactory. The vision depends largely upon the position of the lesions; if the macula escapes it may be quite good and even entirely normal.

Treatment.—This depends upon the cause. If it is syphilis, inunctions of mercurial ointment should be prescribed, to be followed by iodid of potassium, or mercury may be given by the mouth in the form of the protiodid, or by the hypodermic method. Later, a prolonged course of bichlorid of mercury combined with tincture of iron is advisable. *Salvarsan* and *neosalvarsan* or their equivalent—arsphenamin—in the later states of choroiditis have not seemed to the author to be of much value; in acute forms their action is more satisfactory (compare page 346). Subconjunctival injections have been recommended. They may be composed of bichlorid of mercury (1 : 2000–4000), cyanid of mercury (1 : 5000), or physiologic salt solution. Pilocarpin sweats may be tried, and in non-syphilitic cases their effect is sometimes strikingly favorable; in old cases strychnin and the galvanic current have been advised. If tuberculosis is the suspected or definitely established cause, *tuberculin* should be administered (see page 352). Certain cases of intra-ocular tuberculosis (*choroiditis*), especially characterized by chronicity of the lesions, are amenable to this treatment. All close work must be forbidden; the eyes should be protected by dark glasses. Should a mydriatic be employed in the treatment of acute choroiditis, its effect on the intra-ocular tension must be carefully watched and frequent tonometric tests are advisable. Dionin is of service. Naturally, general medication should be governed by the probable etiologic factors, and what has been written on page 366 applies to the disease now under discussion, and in acute choroiditis, especially of the circumscribed variety, great care should be exercised in a search for focal infection, particularly in the buccal mucous membrane, the teeth, tonsils, and intestinal tract.

Central choroiditis is the name applied to choroiditis confined to the region of the macula; its manifestations are numerous.

There may be an irregular patch of exudation, semi- or completely atrophic, and bounded by pigment. This is recognized by the ophthalmoscope, and by a scotoma in the field of vision. Occasionally the area

consists of an epithelial atrophy, either with a well-marked border, somewhat irregular in outline, or with a border less sharply marked and with pigment distributed over the surface of the defect. Sometimes the lesions consist of areas of yellowish exudation, interspersed with small round and linear pigment masses and dot-like hemorrhages. *Hemorrhagic central retinochoroiditis* in non-myopic eyes, characterized by an oval or round area of exudation, surrounded by hemorrhage, is not uncommon.

Again, the macula may be occupied or surrounded by a large white patch, the rest of the eye-ground being normal. Occasionally the area is entirely circular and the deep vessels exposed, or they may be atrophied and converted into white lines (*sclerosis of the choroidal vessels*).

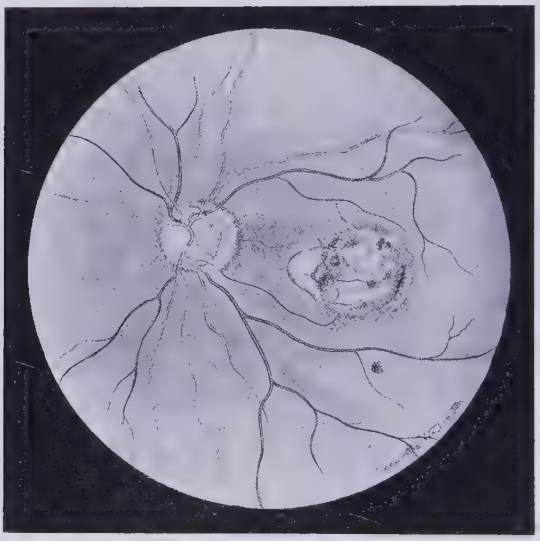


FIG. 174.—Central atrophic choroiditis; on the temporal side of the disk there is a semi-atrophic area, the so-called conus (from a patient in the Philadelphia General Hospital).

Pigment is usually absent. To these types of choroidal change the name *senile areolar atrophy of the choroid* is usually applied. In some cases, owing to atrophy of the pigmented epithelium and the choriocapillaris, there is wide-spread exposure of the larger vessels of the choroid, which, white streaked and sclerotic, constitute a striking ophthalmoscopic picture (*primary sclerosis of the choroid*). Seen in elderly people it may be regarded as a senile degeneration; it has also been attributed to syphilis and to arteriosclerosis. Extensive *sclerosis of the choroidal vessels*, which are apparently converted into white lines, with some pigmentation in the periphery of the eye-ground, but with normal disks and retinal vessels, has been reported as a *family disease*, that is, it may occur in several members of the same family. A form of choroiditis especially described by Förster is known as *choroiditis*

areolaris and affects the region of the disk and the macula. The spots are numerous, but larger than those in disseminated choroiditis. Their centers may be white, and are usually black rimmed and sometimes undermined. A peculiarity of this type of choroiditis is that the foci, first appearing in the macular region, in successive stages gradually approach the periphery where the most recent lesions will be found. Occasionally the macula is covered with a greenish or grayish plastic exudate, of various shapes, often surrounded by a rim of hemorrhage and later by a zone of erosion or atrophy—probably a late stage of one type of plastic choroiditis (see page 391).

In the same region there is observed another variety of the disease, first described by Tay and Hutchinson as central *senile guttate choroiditis*, marked by the appearance of numerous white, glistening dots, somewhat resembling the earlier stages of renal retinitis (Nettleship) and always symmetric, though sometimes an interval of time elapses

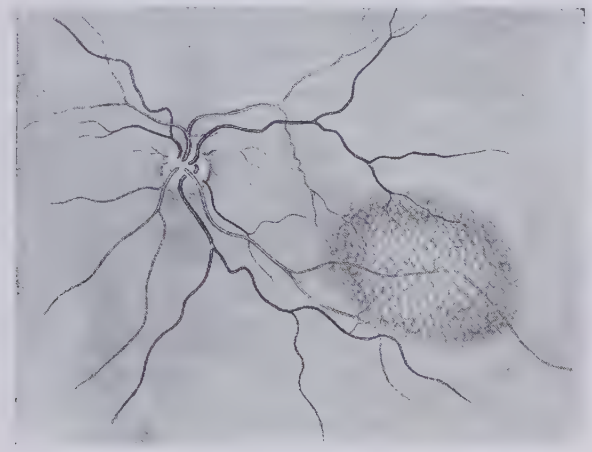


Fig. 175.—Colloid change in the macular region.

before the implication of the second eye. The white spots are due to colloid degeneration and calcareous formations in the choroid and are associated with secondary involvement of the retina. Occasionally the macular region contains an oval or circular patch of dense grayish-white or yellowish-white tissue, which lies beneath the retina and seems to be in the choroid, and which, according to Nettleship, belongs to this group of central senile choroidoretinitis. Usually there are contraction of the field of vision and negative scotoma.

Large areas of *colloid change* may also occur without disturbance of vision (*verrucosities of the choroid*, Fig. 175); also disseminated colloid lesions. Indeed, widely disseminated yellowish spots, existing either singly or in small clusters, are not infrequently found in the eye-grounds of elderly persons, due probably to colloid changes in the lamina vitrea. Vision is usually not disturbed in the absence of macular involvement.

It is important, if possible, to recognize all forms of central choroiditis before a cataract operation is performed. They may be suspected if there is imperfect central fixation for light, but really can be positively determined only while the cataract is still incipient and ophthalmoscopic examination is possible.

Causes.—Central choroiditis of inflammatory type may be caused by syphilis and also by blows upon the eye. Chronic atrophic choroiditis in this region is seen in myopia, and Gould described macular choroiditis as the result of uncorrected ametropia and insufficiency of the internal recti muscles, even in non-stretching eyes (*ametropic*



FIG. 176.—Central cone-shaped lesion in choroid, surrounded by lines of edema (patient in the University Hospital).

choroiditis). Nettleship taught that central senile choroidoretinitis, in its various manifestations, depends chiefly upon disease of the posterior ciliary arteries, either the trunks themselves or the branches which perforate the sclera near the optic nerve, and that any affection of the retina itself is secondary.

Treatment.—In the syphilitic variety the usual remedies are indicated. In types connected with refractive error the best possible correction should be given and absolute eye-rest enjoined. In the senile varieties, both the ordinary and the guttate types, treatment appears to have no influence.

Unclassified Forms of Choroiditis.—In addition to the diseases of the choroid which have been described others appear which cannot be definitely classified:

Large patches of atrophy not located in special portions of the choroid, resulting probably from the absorption of former hemorrhages, or, perhaps, tuberculous areas; *hemorrhagic choroiditis* occurring, as was pointed out by Hutchinson, especially in young men, and resulting in numerous spots of atrophy which are not readily distinguished from those of the syphilitic variety; yellowish or other spots of choroidal disease, which have been attributed to the action of intense light or the effect of heat; slight macular changes in the form of small yellowish or maroon-colored spots, sometimes with a few scattered pigment granules in the immediate vicinity of the fovea, which do not affect vision and are unnoted by the patient. The last-named lesions have

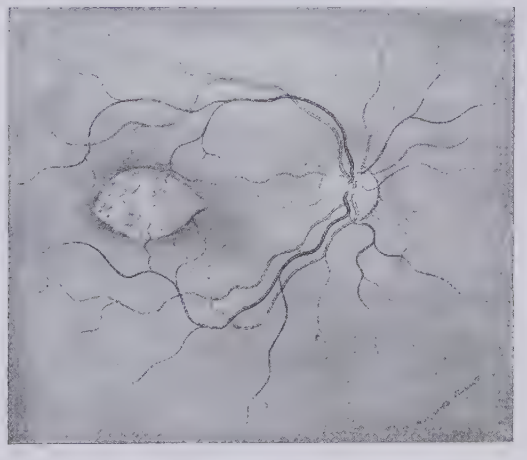


Fig. 177.—Macular coloboma (compare with Fig. 174).

been attributed by some authors to the influence of abnormal refraction, but are sometimes seen in association with transient albuminuria, and probably represent small spots of degeneration due to vascular disease, perhaps of the short posterior ciliary arteries which supply the region of the macula. Occasionally, isolated circumscribed masses of black pigmented material are observed, the fundus oculi elsewhere being normal. Unless situated in the macula they do not affect vision.

Myopic Choroiditis.—Atrophy of the choroid, commonly of a local character, occurs in severe or, as it has sometimes been called, *malignant* myopia, and is observed either in connection with or surrounding the nerve head. It is caused by the elongation which occurs at the posterior pole of the eye, and receives the name *posterior staphyloma;* if the disk is entirely surrounded by the area of atrophy the name *annular posterior staphyloma* or *circumpapillary atrophy* is suitable (Fig. 178).

The term *scleroticochoroiditis posterior* is also applied to this variety of choroidal change, just as *anterior scleroticochoroiditis* is the name given to an inflammatory affection which attacks circumscribed portions of the anterior part of the choroid, with corresponding portions of the sclera, and which, in aggravated instances, may give rise to staphylomatous bulging and gradual loss of vision from opacity of the vitreous and cornea (see page 325). Hirschberg objects to the term "myopic choroiditis," as he believes the lesions are mechanical and not inflammatory in origin.

Semi-atrophic and *atrophic crescents* (often inaccurately called "conus") also appear at the outer margin of the disk in astigmatic eyes, and in eyes undergoing change owing to the distention of their coats from too close work aggravated by imperfectly or improperly corrected errors of refraction. In hyperopic and emmetropic eyes narrow white crescents, usually at the temporal side of the disk, are often evident. These are the so-called *scleral crescents.*

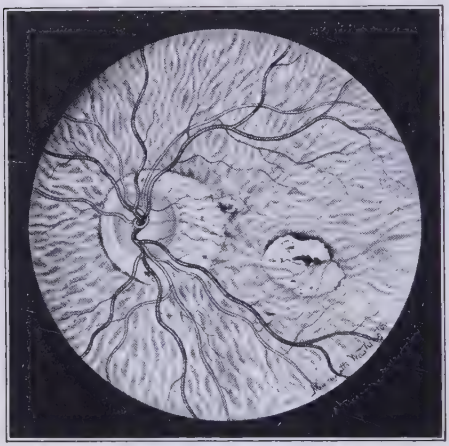


Fig. 178.—Myopic choroiditis. The cut ilustrates posterior staphyloma—the whiter area surrounding the nerve: atrophic choroiditis in the macula—the white patch bordered by pigment in the central part; and general exposure of the choroidal vessels by absorption of the retinal pigment epithelium.

In the macular region in myopia there may be very decided semi-atrophic or atrophic patches having the general characteristics of the spots already described, and greatly interfering with vision. The process begins in the form of small rents which gradually coalesce into an atrophic patch. In like manner this area may be involved by a hemorrhage in progressive myopia, which after absorption leaves impaired vision, owing to the damage of the overlying retina. The vessels of the choroid are exposed by maceration and absorption of the retinal pigment epithelium, causing the appearance described under superficial choroiditis (see Fig. 178, and also page 387).

Suppurative Choroiditis and Iridochoroiditis (*Endophthalmitis* [Fuchs]).—Acute iritis, which occasionally complicates inflammation of the choroid (see page 341), and the chronic type of iridochoroiditis, which tends to loss of vision and shrinking of the eyeball, have been described (see pages 354 and 364).

The present disease, however, is distinguished by a suppurative process which may begin in the choroid, or in the vitreous, or in the aqueous chamber and pass into the vitreous. Fuchs classifies *endophthalmitis* thus: the purulent exudation remains confined to the posterior

part of the eye (*abscess of the vitreous*), or extends from the posterior region to the anterior chamber, or begins in this chamber (*purulent iritis*), or the whole interior of the eye is involved (*panophthalmitis*).

Symptoms.—If there is sufficient transparency of the media, and in simple abscess of the vitreous there may be at first only slight or no exterior manifestations, a mass of exudation may be seen behind the lens in the vitreous, giving rise to a yellowish reflection when viewed by transmitted light, or often visible to the unaided eye (*pseudoglioma*, "amaurotic cat's eye"; see also page 470). At first the tension may be raised and the anterior chamber shallow; later the tension is lowered; the pupil is dilated; vision is lost, although light perception may at first be present.

If the process remains localized in the vitreous the inflammatory symptoms subside, the intra-ocular tension lessens, membrane formation takes place, and the eyeball gradually shrinks (*atrophy of the eyeball*). If the process passes forward into the aqueous chamber or begins there, there are suppurative inflammation of the iris, haze of the cornea, turbidity of the aqueous, and exudation in the pupil area. Pain increases and becomes severe. The eyeball may ultimately shrink or the inflammation may spread to all of the interior ocular coats and the edema of the lids and chemosis of the conjunctiva are intense, the pain violent, and the constitutional symptoms—fever, chills, nausea, and vomiting—are very marked. The inflammation involves Tenon's capsule and causes protrusion of the globe, which is pressed against the swollen lids until these can scarcely be separated. Finally, rupture of the sclera or sloughing of the cornea occurs, the purulent matter finds a vent, the pain subsides, and in about six weeks the ball is soft, sightless, and shrunken. The second outcome of purulent choroiditis is known as *panophthalmitis*, and the ultimate result is *phthisis bulbi*.

Causes.—*Suppurative choroiditis*, or *iridochoroiditis*, may be due to an infection that comes from the outside, that is, an *exogenous infection*. In these circumstances it may arise as the result of perforating wounds and injuries, operative wounds which have become infected—for example, cataract extraction—sloughing ulcers and abscesses of the cornea, and prolapse of the iris. It may be due to thin, permeable cystoid corneal cicatrices and to corneoscleral trephining, a diminutive aperture having formed in the delicate conjunctival covering, permitting the entrance of micro-organisms.

Suppurative choroiditis may also be caused by embolism from a focus of suppuration (*endogenous infection*), and produces the condition which is known as *metastatic ophthalmitis*. From the etiologic standpoint, following Axenfeld's classification, metastatic ophthalmitis may result from puerperal pyemia, which is its most frequent cause; from surgical pyemia, which includes all cases which arise from injury, operations, and local purulent areas, even where the last-named conditions are non-traumatic, but have an internal origin, and may have their situation in the mucous membrane of the digestive, pulmonary, and urinary organs (Groenouw); from cryptogenetic septicopyemia,

that is, the point of entrance of the infection has not definitely been determined, and, finally, from infectious diseases, particularly pneumonia, influenza, measles, scarlet fever, diphtheria, and smallpox. The disease may also result from cerebrospinal meningitis, basic meningitis, dysentery, bronchitis, whooping-cough, inflammation of the umbilical vein, and thrombosis of the orbital veins. It may be bilateral or unilateral, and the puerperal cases usually develop during the first two weeks of the disease, but may be delayed until the seventh week. Ulcerative endocarditis is a frequent complicating factor.

Pathology.—Examination of eyes in which suppurative choroiditis has occurred shows the presence of a thick purulent infiltration of the choroid, involvement of the overlying retina, and sometimes conversion of the entire vitreous into a purulent material. In the metastatic variety of the affection the septic masses enter into the capillaries of the eye. Sometimes the retina is exclusively or, at least, first affected, later the uveal tract is also involved.

Fuchs' investigations of the anatomic changes in inflammation of the choroid and those which result from infection of the vitreous indicate that the inflammation spreads to the inner lining of the vitreous, especially to the *pars ciliaris retinæ* and to the retina itself. Purulent retinitis results, and the choroid is seriously involved where the inflamed retina remains in contact with it (*endophthalmitis septica*).

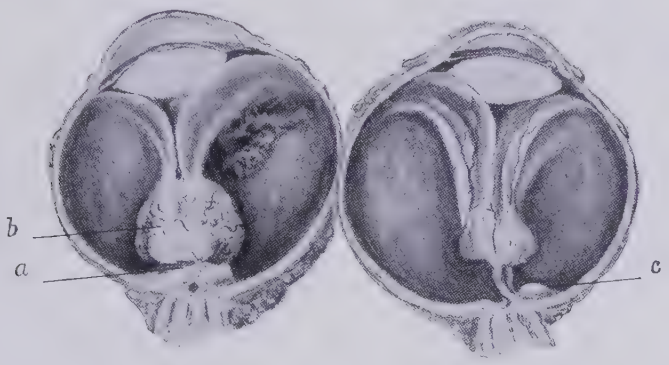
Streptococci, staphylococci, and sometimes Fränkel-Weichselbaum pneumococci have been found, and in many cases of panophthalmitis not necessarily metastatic in origin special bacilli are present, some of which have been determined to have pathogenic significance. This is particularly true after injuries of the eye, and in a number of instances those organisms have been found to which Haab has given the name *panophthalmitis bacilli*, and which belong to the group of the "hay bacilli."

Prognosis.—This is most unfavorable, and almost invariably a shrunken eyeball is the result of the inflammation. A few cases of recovery from suppurative iridochoroiditis following cerebrospinal meningitis have been recorded. The termination of destructive ophthalmitis in children is usually not fatal, but a few deaths have occurred, generally from meningitis (see also page 471). In bilateral cases of puerperal metastatic ophthalmitis the mortality is exceedingly high, only a few authentic cases of recovery being on record.

Treatment.—Copious irrigations with the usual antiseptic lotions and with a solution of sulphate of magnesium (page 225) and frequently changed ice compresses are useful. In later stages hot fomentations are sometimes more agreeable, a square of lint soaked in heated bichlorid solution being applied to the eye; and opium and quinin in full doses are indicated. If there is much pain before spontaneous rupture has occurred, a free incision into the sclera may bring relief. The methods of treatment to prevent the spread of septic processes after injury have been described on page 332, and other methods of treatment will be found on page 471.

Surgeons differ in regard to the advisability of enucleating the globe during the acute stages of panophthalmitis, some operators declining to perform excision in such circumstances, in the belief that meningitis is liable to follow, while others do not recognize this danger.

The author does not hesitate to enucleate an eyeball in which there is suppuration if the surrounding orbital tissues are not yet involved in the process; but agrees with Pooley that where the process has reached a great height, where there is purulent infiltration of the orbital tissues, and where the affection has begun posteriorly, as in some varieties of septic iridochoroiditis, the operation of enucleation is surrounded by dangers. In a certain number of cases enucleation has been followed by meningitis once in the author's experience, the patient being a diabetic subject. Therefore under these conditions evisceration is preferred. But even after evisceration there may be a great accumulation of inflammatory products behind the scleral cup, and to these a vent must be given.



FIGS. 179, 180.—Leukosarcoma of choroid, showing at *a* constriction which marks where choroidal capsule was ruptured and where retina became adherent, being pushed forward with growth of upper part of tumor (*b*), which assumes a mushroom shape. At *c* choroidal origin of growth is seen.

Tumors of the Choroid.—The most frequent neoplasm of the uveal tract and, for the present purposes of description, of the choroid is *sarcoma*. Most commonly it appears as a pigmented growth (*melanosarcoma*); more rarely (1 in 8, according to W. C. Rockliffe) without pigment (*leukosarcoma*).

Sarcoma of the choroid, according to E. Pawel, is most frequent between the ages of fifty and sixty. A good many cases, however, occur at an earlier period than this, but sarcoma is of rare occurrence prior to the twentieth year. One patient in the author's experience with melanotic sarcoma of the choroid was only fourteen years of age. Men are more frequently affected than women, and the left eye, according to some statistics, is more apt to be involved than its fellow.

Pathology.—The growth usually is circumscribed, and has a spheroid form as long as the choroidal capsule remains unbroken. Sometimes it assumes a cake-like shape, and occasionally the form of a

mushroom. Rarely there is diffuse sarcomatous infiltration of the choroid.

Sarcoma of the choroid is almost invariably a primary growth, but the choroidal coat may be, though very rarely, affected by a metastasis occurring from a tumor in some other portion of the body—for example, the mediastinum (A. V. Meigs and the author, Wiener).

The tumor develops from the outer layers of the choroid, and grows inward, detaching the retina. The cells are round or spindle-formed or occasionally of a large endothelioid type, provided they develop from the endothelial linings of the lymph-spaces. They are usually pigmented, the density of the pigmentation depending upon the participation of the choroidal stroma cells in the proliferative process. Usually there are many broad vessels around which the cells may be grouped. Intra-ocular sarcomas may undergo necrosis from insufficient oxygenation owing to the death of groups of cells at a distance from blood-



Fig. 181.—Macroscopic appearance of a pigmented choroidal sarcoma—flattened growth or so-called cake-like form. One extrascleral nodule.

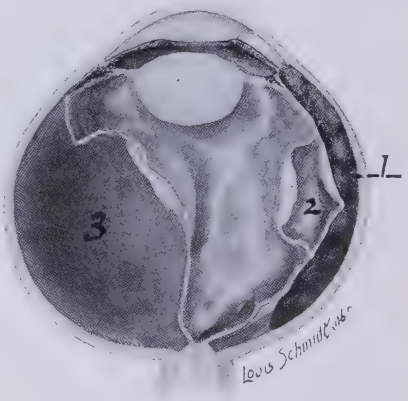


Fig. 182.—1, Pigmented flat sarcoma; 2, cyst of retina; 3, detachment of retina in portion of eye opposite to position of tumor.

vessels. Toxins are liberated which devitalize the surrounding cells (Fuchs). In the second stage secondary glaucoma occurs, and occasionally plastic iridocyclitis appears and results in atrophy of the globe. A tumor may grow in a phthisical eye, and, as Leber pointed out, an eye which is the seat of a growth may become phthisical and the tumor itself cease to grow for a time. Coppez divides the primary new growths of the choroid into—(1) *interfascicular endotheliomas* which develop from the *endothelial* cells of the lymph-spaces; (2) *peritheliomas* (angiosarcomas) which arise from the perithelial cells of the blood-vessels; (3) *sarcomas* of various characters which grow from the proper cells of the choroid and the adventitia of the blood-vessels. *Alveolar sarcomas*, also called endotheliomas or intravascular angio-

sarcomas, are rare as compared with perivascular sarcomas of the choroid. It is probable that in these alveolar types the greater mass of the tumor is formed by the proliferation of endothelial cells.

Diffuse sarcomas of the choroid are classified by Parsons into two subgroups—*flat sarcomas* and *ring sarcomas*. They are characterized by an infiltrating tendency, as opposed to the formation of a definite tumor. They exhibit large round or polygonal cells, alveolar or plexiform arrangement, hyaline and myxomatous degeneration, and extension along the perivascular lymph-spaces. According to Parsons, they should be considered as endotheliomas, and they spring from the lining cells and proliferate in the spaces which they line. The onset of glaucoma is early in diffuse sarcoma.

Parsons has also called attention to certain *anomalous sarcomas of the choroid* which bear a microscopic similarity to organizing blood-clot



FIG. 183.—Pigmented sarcoma at *a*; retina detached and folded.

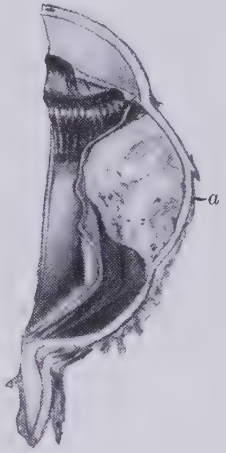


FIG. 184.—Leukosarcoma at *a*; retina detached.

and are of comparatively low malignancy. According to him, if they are not excised, they shrink, and represent the tumors before referred to which have been found in phthisical eyes. Destructive hemorrhage may occur in choroidal sarcomas, as has been specially pointed out by Verhoeff. Hemorrhages of this character may be responsible for the sudden attacks of glaucoma which occur in choroidal sarcoma. *Melanoma of the choroid*, that is, a small circumscribed mass of chromatophores (usually only accidentally found in microscopic examination) may be the origin of a sarcoma, as has been reported, for example, by the author and E. A. Shumway. The smallest sarcomas on record have been described by Fuchs (in one instance the growth being between 0.7 and 0.8 mm. in the horizontal and vertical diameters), and Shumway and the author have also observed a very small sarcoma of the choroid (4.4 mm. in the anteroposterior diameter and 0.9 mm. in thickness).

Symptoms.—The life history of a sarcoma of the choroid has been divided by systematic writers into four periods: The first, the quiet period; the second, the glaucomatous period; the third, the extra-ocular period, or that stage when the growth bursts through the scleral boundary; and fourth, the period of metastasis.

In the *first stage* the disease resembles a detachment of the retina, this membrane being pushed forward by the underlying elevation, the whole being surrounded by a serous effusion. Beneath this retinal covering the brownish mass may sometimes be recognized, covered by irregular choroidal vessels, except in the non-pigmented varieties. If the growth is situated far forward, it is sometimes possible to examine it by means of oblique illumination through a dilated pupil. There is a corresponding defect in the field of vision, and the sight of the affected eye is diminished in accordance with the situation of the tumor. Should this be peripheral, the central vision at this stage may not be seriously disturbed. The first stage usually lasts from six to twelve months, but rarely may be prolonged to five years. Occasionally sarcoma of the choroid produces symptoms resembling *tenonitis* (Kipp and the author).

In the next period of the history of this growth, or *glaucomatous stage*, symptoms of increased tension which depend upon alterations in the angle of the anterior chamber arise: pain in the brow, anesthesia of the cornea, shallowing of the anterior chamber, and dilatation and tortuosity of the perforating episcleral vessels. Ophthalmoscopic examination is no longer possible, the localized detachment of the retina becomes general by increased serous effusion, the lens may become cataractous, and a severe iridocyclitis may supervene. Glaucoma, however, as noted above, may develop in earlier periods than the one just described.

As the growth continues, the sclera is ruptured and the surrounding tissues are involved (*fungous state* or *stage of episcleral tumors*). It may pass backward into the brain, or secondarily affect the optic nerve, but more commonly the last, or *metastatic stage* (*stage of generalization*), develops; distant organs are attacked by growths of similar histologic character, the liver far more frequently than other organs, but also the spleen, intestines, and even the lungs. Metastasis to the liver need not necessarily be delayed until the tumor has burst, at least visibly, through the scleral boundary. The most extensive secondary sarcoma of the liver which has come under the writer's notice developed from a small sarcoma of the choroid apparently entirely confined within the scleral covering.

Diagnosis.—It is necessary to differentiate sarcoma of the choroid from glioma of the retina. (See page 535.)

In the early stages choroidal sarcoma may be mistaken for ordinary detachment of the retina, for detachment of the choroid, or for a subretinal exudation as has been reported by A. Knapp and H. Friedenwald. In retinal detachment there is usually a history of sudden onset, and the ophthalmoscope may reveal undulations of the folds of the

detached retina, vitreous opacities, and signs of choroiditis. Moreover, the field is frequently less sharply defective than in choroidal sarcoma, although this is not an especially trustworthy sign. *Rents in the retina*, so commonly found in simple retinal detachment, do not occur in separation of the retina due to tumor (Lister). Pigment spots are often visible beneath the retina which overlies a sarcoma of the choroid. Inasmuch as early detachment of the retina occurs in many cases of sarcoma of the choroid, all apparently simple detachments of the retina should be most carefully studied, especially by means of *transillumination (diaphanoscopy)*. Various instruments have been designed, notably those of Leber, Sachs, and Würdemann. The eye having been cocainized, the point of the instrument is passed over all areas of the exposed sclera. In the absence of a growth the red glare in the pupil remains undisturbed and bright; if a growth exists, the passage of the light is obstructed as the point of the instrument is placed over the region beneath which it is situated, and the pupil remains dark. According to Griscom the transillumination test applied to eyes densely pigmented (those of negroes, for example) is not satisfactory. In order to detect tumors situated far posteriorly Lancaster has mounted a small Tungsten lamp at the end of a flexible copper tube attached to a posterior flashlight battery. Through an incision in the conjunctiva and capsule of Tenon the lamp can be carried behind the eyeball and its posterior segment thus transilluminated. Investigation of the eye with the aid of the Gullstrand diaphragm lamp, whereby a brilliant beam of light is thrown into the pupil, while the surgeon also looks through it with an ophthalmoscope and observes the color of the mass, black or reddish, according as it is opaque or translucent, is called *pupillary transillumination*. The value of *ophthalmodiaphanoscopy* in these circumstances has been described on page 119.

Choroidal detachment is rare, except as it may be caused by operation (see page 410); the history is different from that of sarcoma, and the characteristic vessels of the choroid can usually be recognized beneath the vessels of the retina.

Too much reliance cannot be placed upon the tension of the eyeball as a distinguishing sign between sarcoma and retinal detachment, because intra-ocular tension may be unaltered in each instance, although, as C. Devereux Marshall has shown, it is probably never diminished (as it often is in retinal detachment) in undoubted cases of choroidal sarcoma, while it may be reduced in cases of sarcoma of the ciliary body.

In the stage of increased pressure the disease may be distinguished from glaucoma by the history of the case, by testing the eye with a transilluminator, and by instilling a miotic, which in ordinary acute glaucoma should be more effective than in an eye containing a sarcoma in the glaucomatous stage. An eye in the state of absolute glaucoma should always be carefully examined in view of the fact that it may contain a morbid growth.

Pseudotumors of the uveal tract should be considered in the study of choroidal growths, as choroidal and retinochoroidal exudation, sub-

retinal exudation, and massive retinal exudation have been mistaken for sarcoma. Repeated examinations are required to decide the diagnosis.

Prognosis.—Removal of an eye for choroidal sarcoma results in a cure in from 25 to 30 per cent. of the cases, although statistics on this point vary greatly. Hirschberg's published statistics may be briefly summarized as follows: Local recurrence, 2.5 per cent.; metastasis, 41.5 per cent.; cure, 56 per cent. He points out that statistics show a steady improvement in so far as permanent recovery after enucleation for sarcoma of the choroid is concerned. His own earlier operations yielded only 35 per cent. of recoveries. Metastasis to internal organs is the most usual cause of death and may take place within two years after operation or may be delayed to a later period. The stage at which enucleation is performed does not certainly influence the occurrence of metastasis, although, as Hirschberg maintains, *operation at the very*

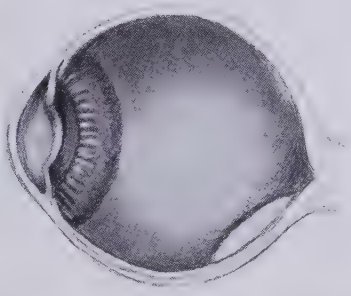


Fig. 185.—Metastatic carcinoma of the choroid.

earliest stage should be urged. A recent statistical inquiry by von Hippel (132 cases followed up for more than five years) failed to demonstrate that metastases were more frequent after operation in the second stage than after those performed during the first stage. He also, however, advises *early* enucleation, being willing to postpone it only in case the patient has but one eye which retains useful vision. It is usually stated that very vascular and round-celled sarcomas are more fatal than other varieties. Prognosis is better in young than in aged subjects. Local recurrence is much less frequent than metastasis; it is prevented by prompt removal of the eye. If there is no recurrence or metastasis within four years after enucleation of the eye, this complication becomes unlikely, although exceptions to this rule have occurred, and metastasis has been noted even after seven years.

Treatment.—From what has been said it is evident that the only treatment is prompt enucleation. The optic nerve should be severed as far back in the orbit as is possible. It may be necessary to remove the entire contents of the orbit. The treatment of choroidal sarcoma

by radium has not yielded very satisfactory results; but radium should be applied to the socket after enucleation.

Rare forms of tumor of the choroid are the following: *Cavernous angioma, telangiectatic sarcoma, adenoma,* and *enchondroma.* Angiomas of the choroid (about 21 are on record) may occur in connection with nevi of the face.

Carcinoma of the Choroid.—The tumor (a comparatively rare growth, about one-third being bilateral) is of rapid development and generally appears as a flat growth in the neighborhood of the macula of yellowish or pale gray color; pigment dots may or may not be present on the swelling (Usher). The average duration of life according to Usher's investigations after the eye is affected is practically eight months; the longest time recorded is two years; the shortest, one month In the majority of instances it represents a metastasis from a carcinoma of the mammary gland (39 times in 64 cases collected by Suker and Grosvener, 65 times in 90 cases by Usher); the primary neoplasm has also been situated in the lungs, pleura, stomach, liver, thyroid, esophagus, mediastinal glands, suprarenal gland, prostate, and ovary. The metastasis takes place through the posterior ciliary arteries and later develops in the perivascular lymph spaces.

Tubercle of the Choroid.—Tubercles appear in the choroid as yellowish-white spots, varying in size from 1 to 1.5 mm., occasionally larger, and usually, though not necessarily, associated with similar growths in the meninges (tuberculous meningitis). Repeated examination is required for their detection, and even then they may escape observation owing to their diminutive size (*choroidal dust*). The facility of their detection has been much enhanced since the introduction of electric ophthalmoscopes. They have been frequently found in post-mortem examinations.

Tubercles, known as miliary tubercles, are distinguished chiefly by their color, which has been described as of a dull yellowish white in the center, encircled by an ill-defined rose-colored area (Horner). Usually there are no pigmentary changes in the immediate neighborhood, but pigment bodies may surround the nodules if they are prominent (Bach). They are situated usually near the optic disk or in the macular region, and vary in number from three to six or many more. Optic neuritis and tubercles in the choroid may develop at the same time during meningitis.

Instead of the miliary growth, a single large *tuberculous tumor* may appear and progress, producing the same destructive changes as a sarcoma. It may be associated with a similar one in the brain.[1] According to Zur Nedden, the age of patients suffering from tuberculous tumor of the choroid has varied between one and a half and sixty-two

[1] *Chronic choroidal tuberculosis* is characterized by optic neuritis, optic atrophy, hemorrhages (tuberculous inflammation), and a diffuse, yellowish-white discoloration, occupying a considerable area of the eye-ground, within which are round, yellowish-white spots. Michel described tuberculous granulation tumors of the choroid, which began with the appearance of retinal detachment, and later caused abscess in the vitreous and shrinking of the eye.

years, although the age of childhood has furnished by far the greatest percentage. The condition, especially if the growth has attained a definite size, must be differentiated from glioma of the retina in the young and sarcoma of the choroid in adults. The evolution of conglomerate tubercle of the choroid is usually more rapid than that of tumor. Scleral involvement and perforation generally occur early in the disease. Choroidal tuberculosis is rarely primary. Other signs of tuberculosis are usually found in the general system. Occasionally in eye-grounds otherwise normal, and in patients in good health, isolated areas of choroidal atrophy with or without pigment heaping, or large patches of dense, slightly elevated pigment are seen, which probably represent healed tuberculous lesions—the so-called *obsolescent*

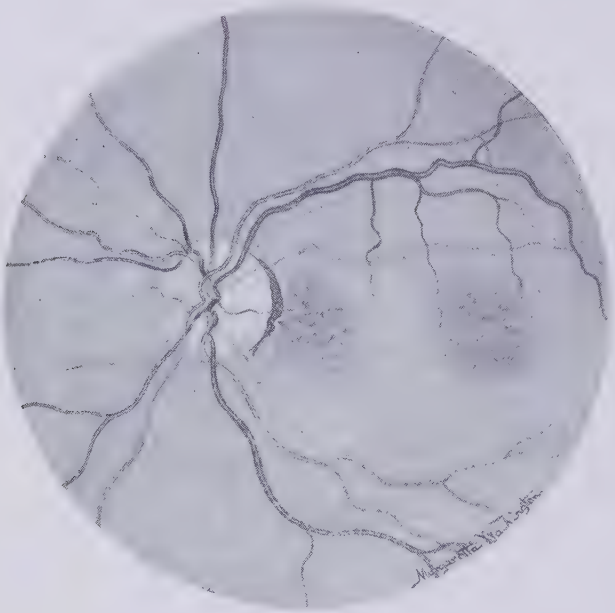


FIG. 186.—Tubercles in the choroid.

tubercles or tuberculous areas. The relation of tuberculosis to choroiditis has been elsewhere described (page 392).

Treatment.—Miliary tubercles of the choroid do not require any treatment directed to the eye itself, the vision of which may not be seriously affected. If a single large choroidal tumor is recognized, and the patient's general condition permits it, enucleation to avert general tuberculosis has been suggested. Instead of surgical procedures, injections of tuberculin (T. R.) have been employed by von Hippel and others with encouraging results, and should be given full trial (see also page 352).

Injuries of the Choroid.—Wounds of the Choroid.—Necessarily, in a perforating wound of the sclera, the choroid is also lacerated

or incised, and no description other than that already given in this connection is required.

Foreign Bodies in the Choroid.—A foreign body, usually metal, may lodge in the choroid; the treatment has been described on pages 328 and 329.

Rupture of the Choroid.—The most important injury to which the choroid is subject, and which follows a blow upon the eye, is rupture. This generally manifests itself in a sickle-shaped crescent, commonly on the temporal side of the disk, rarely on the nasal side, and which may extend in a horizontal direction. The rupture may be single or multiple, and sometimes is composed of several branches. The immediate effect of the blow is a hemorrhage preventing distinct observation. After its disappearance the fissure is evident to the ophthalmoscope as

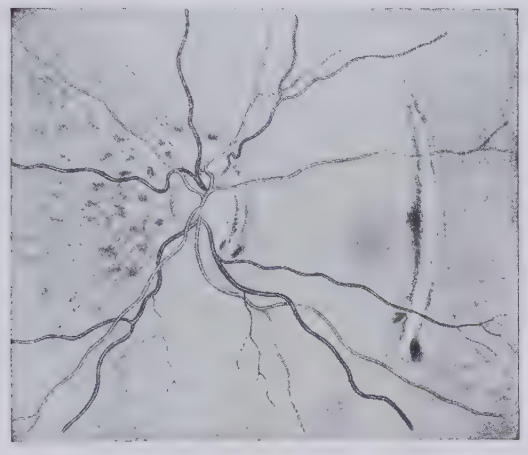


FIG. 187.—Rupture of the choroid on the temporal side of the disk and pigmented traumatic choroiditis (see page 392) on the nasal side (from a patient under the care of Dr. Randall in the Children's Hospital).

a yellowish-white stripe bordered with some disturbed pigment (Fig. 187). (See also page 683.)

The ruptures usually run concentrically with the papilla.

They may be either complete or incomplete, and may or may not be associated with breakage of the overlying retina. In rupture confined to the choroid the retinal vessels pass over it. If the retina has also given way there is apt to be more hemorrhage than without such accident, and no retinal vessels are observed crossing the choroidal separation. Associated with choroidal rupture there may be a rupture of the sphincter of the iris (Duane and the author).

The ultimate effect of vision depends upon the size and situation of the rupture. At first there is very considerable disturbance of sight, partly due to effusion and partly to injury of the iris, sometimes associated with blood in the anterior chamber. This slowly clears away, and very good vision may result provided the change in the eye-ground

has not been extensive. A deterioration of vision may occur a long time after such an injury owing to secondary changes in the optic nerve.

Treatment.—The pupil should be dilated with atropin; if there is much pain, a leech or two should be applied to the temple, a pressure bandage adjusted, and the patient put to bed. These measures suffice to encourage the absorption both of the blood and of the serous effusion.

Hemorrhage Into the Choroid.—In the section of Unclassified Forms of Choroidal Disease variously shaped hemorrhages which appear in this membrane, and which by absorption give rise to atrophic spots, have been described. In like manner there may be hemorrhage from the choroid, the result of a blow. A choroidal hemorrhage may be distinguished from one situated in the retina by noticing the more diffuse character of the extravasation and the fact that the retinal vessels pass over it, but the diagnosis is difficult.

Detachment of the Choroid.—This is a comparatively rare clinical condition, although not infrequently found in enucleated, shrunken eyes. It may be spontaneous or traumatic, partial or complete. The detachment may be caused by blood, serum, a layer of lymph, or a new growth. But following cataract extraction, iridectomy for glaucoma, iridosclerectomy, and corneoscleral trephining (see page 740) detachment of the choroid is not very uncommon. The detached choroid protrudes as a dark mass into the vitreous, and the anterior chamber is shallow or obliterated. It is caused by the passage of the aqueous humor through a rent in the attachment of the ciliary body beneath the choroid. The prognosis generally is favorable.

Meller, who has carefully studied the subject, classifies detachment of the choroid thus: Early postoperative detachment, innocent in character; late postoperative detachment, following trauma or occurring without cause; spontaneous detachment, either innocent, resembling postoperative separation, or malignant, causing blindness. If the retina is detached with the choroid the prognosis is less favorable.

Ossification of the Choroid.—This is occasionally found in eyes long blind and shrunken from destructive iridochoroiditis. The formation of bone occurs in the inflammatory tissue, and may be recognized by palpation in the form of an irregular plate, spicule, or complete shell. *Calcareous degeneration* is common in eyes of this character. The eyeball should be enucleated.

Atrophy of the eyeball is a condition characterized by diminution in the size of and by alteration of the shape of the globe, caused by contraction of inflammatory exudations—for example, those formed in the uveal tract, or in the vitreous, followed by detachment of the retina. The eyeball is somewhat quadrate in shape and grooved by the pressing action of the recti muscle. The cornea is small; it may be opaque, but sometimes is quite clear and protuberant. The iris is atrophic. It should be sharply distinguished, as Fuchs points out, from *phthisis bulbi*, which results from a suppurative inflammation (see page 399) and is associated with rupture of the sclera and partial

evacuation of the ocular contents. Such an eyeball should be removed; sometimes it produces sympathetic irritation.

Hypotony, the reverse of elevation of tension (*hypertonia*), occurs under many conditions, to some of which reference has been made. It is conveniently discussed in this place. Diminished intra-ocular pressure is a constant symptom after perforation of a corneal ulcer, in association with a fistulous scar or a corneal fistula, following escape of vitreous as the result of penetrating wounds of the sclera, etc. (page 327). Hypotony may be due to a violent contusion of the eye, without rupture of the ocular coats, but associated with grave internal lesions—vitreous disorganization and hemorrhage, rupture of the choroid, detachment of the retina, etc. It may be a symptom of paralysis of the sympathetic. In *diabetic coma* the eyeball may be soft, usually, but not invariably, a sign of evil prognostic import. It does not occur in simple diabetes or acidosis without coma (Riesman).

Ophthalmomalacia (*essential phthisis bulbi*) is the name applied to a condition of the eye characterized by hypotony (softening) and diminution in its size which may appear spontaneously and is unrelated to inflammation. There may be photophobia, pain, miosis, and drooping of the upper lid. The condition may last for a few hours only or for several days. Usually there is a return to the normal conditions. An intermittent variety has been described. It may follow injury and has been attributed to disease of the sympathetic.

CHAPTER XI

GLAUCOMA

Glaucoma is the name applied to a disease the essential symptoms of which in its various manifestations depend upon increased intra-ocular tension.

Varieties of Glaucoma.—Systematic writers are accustomed to divide glaucoma into (1) *primary glaucoma*, or that form which arises independently of clinically evident antecedent disease of the eye, and (2) *secondary glaucoma*, or that form which occurs as the sequel of a pre-existing ocular disease, often an inflammation of the uveal tract.

It is customary to divide the primary variety of this disease into (1) *acute congestive glaucoma* (formerly called acute inflammatory glaucoma) and (2) *chronic non-congestive glaucoma* (simple glaucoma, glaucoma simplex).

For clinical purposes it is convenient to retain these descriptive titles, but it should be distinctly remembered that in a certain sense the divisions are artificial, because an acute glaucoma may cease to retain its congestive character and manifest signs which indicate the chronic (non-congestive) variety of the disease, and the so-called glaucoma simplex may at any stage of its career develop symptoms of an acute progress, and lose its non-congestive character; also, its congestive phenomena having subsided, it may reassume its non-congestive (chronic) disposition. In other words, acute and chronic glaucoma are fundamentally the same disease which may be interpreted sometimes by acute (or subacute) congestive and sometimes by non-congestive symptoms.

Symptoms.—The following is a syllabus of the symptoms common to the disease glaucoma, although all of these symptoms are not constantly present in each variety of its manifestations.

1. *Rise in intra-ocular tension*, or increased hardness of the eyeball. Formerly variations in the intra-ocular pressure were designated by the symbols T? ("stiffened sclera") to T + 3 ("stony hardness"). Intermediate degrees were expressed T + 1 and T + 2.

Since the introduction of satisfactory instruments, particularly the instrument of Schiötz, the crude estimation of the impressibility of the eyeball by means of finger palpation has given place to the tonometer (page 93); it measures the impressibility of the eyeball from the degree of which the intra-ocular pressure is inferred. In the absence of an instrument of precision finger palpation of the globe must be utilized and the student should by practice train his fingers in this respect.

2. *Change in the Size and Shape of the Pupil and Mobility of the Iris.*—The pupil may be round or it may be oval or egg shaped, with

the long axis vertical; it may be semidilated, or expanded to its fullest limit; the iris is sluggish in movement or entirely inactive.

The pupillary space sometimes transmits a greenish reflex (hence the name given by the older writers) from the surface of the lens. The dilatation of the pupil is explained by paresis of the ciliary nerves or by constriction of the vessels of the iris. Partial atrophy of the lesser circle of the iris, which may lead to permanent dilatation of the pupil, is not uncommon after acute attacks of increased tension (Hirschberg).

3. *Loss of the Transparency of the Cornea.*—The cornea somewhat resembles the appearance of glass, the surface of which has been dulled by being breathed upon. This haziness is marked in the congestive types of glaucoma, but is absent or only slightly present in the non-congestive varieties. If the cornea is carefully examined, the cloudiness will be found more decided in the center, and consists of numerous closely aggregated points, the whole presenting a stippled or "needle-stuck" appearance. Iritis and iridochoroiditis may produce a similar appearance. The condition has been attributed to an edema of the cornea. Loss of corneal transparency with increased intra-ocular tension, such as may be caused, for example, by external pressure on the eye, is due, according to v. Fleishl, to the corneal fibers becoming doubly refracting.

4. *Change in the Depth of the Anterior Chamber.*—This symptom varies from an almost imperceptible shallowing to a complete obliteration. During the course of glaucoma the lens system and peripheral portion of the iris are pushed forward, and this causes the lessening of the depth of the anterior chamber. The two factors on which Elliot lays stress are overdistention of the vitreous body and congestion of the ciliary body, and adhesion of the base of the iris to the corneal periphery.

5. *Change in the Normal Appearance of the Iris and Turbidity of the Aqueous and Vitreous.*—The same edema which affects the cornea may also cause loss in the characteristic markings of the iris, so that its pattern becomes indistinct, especially in congestive forms of glaucoma. The veins of the iris may be dilated and tortuous; small hemorrhages are sometimes visible. Opacities in the media also are liable to develop, and the lens itself may become cataractous.

6. *Alterations in the Conjunctival and Episcleral Vessels.*—In acute glaucoma there are usually general hyperemia and often edema of the conjunctiva, but in chronic congestive and sometimes even in non-congestive glaucoma there are marked enlargement and tortuosity of the episcleral venous branches (see System II, page 50).

7. *The Excavation of the Nerve-head and the Surrounding Yellowish "Halo," or "Glaucomatous Ring."*—Under the influence of increased intra-ocular pressure the lamina cribrosa recedes, the optic nerve-fibers also give way, the surface of the nerve-head sinks below its normal level, and the *glaucoma-cup* is produced. Sometimes prior to the development of a "cup" a delicate surface depression of the disk is visible, which is highly suggestive of coming events. According to Knies, congestion and edema of the nerve-head precede cupping, and according to Brailey

and Edmunds actual neuritis appears in advance of increased tension. The author and Gasparrini have seen glaucomatous excavation of the papilla follow retrobulbar neuritis. Wahlfors denies that increased intra-ocular tension alone is sufficient to cause cupping of the nerve-head, inasmuch as it requires a pressure of 135 mm. of mercury to produce such an excavation, and in glaucoma the rise rarely exceeds 100 mm. According to him, atrophy of the choroid is an important factor in this respect, because the resistance of the lamina cribrosa is thus reduced, owing to interference with the vessel-bearing tracts which pass from the surrounding choroid into the nerve-trunk and branch in the anterior layers of the lamina. According to Schnabel, the excavation in the nerve-head in glaucoma does not depend upon increased intra-ocular tension, but upon a form of degeneration of the optic nerve-

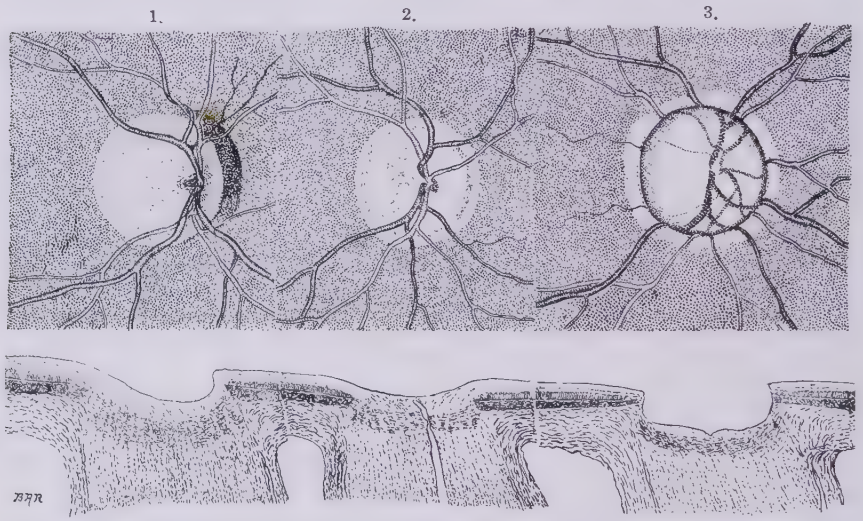


FIG. 188.—Excavations in nerve-head: 1, Physiologic; 2, atrophic; 3, glaucomatous excavations. (From a drawing by Randall.)

fibers, which causes their complete disappearance and the formation of small cavities, both anterior and posterior to the lamina cribrosa (*cavernous atrophy of the optic nerve*). *Lacunar atrophy of the optic nerve,* however, has also been observed in myopia without hypertony and is often not found in eyeballs which have long been the subjects of increased tension. There is no doubt that cupping of the nerve-head in glaucoma should be attributed to the direct influence of the increased pressure within the eyeball.

As the result of release of increased intra-ocular tension a glaucomatous cup may partially or even completely disappear. This observation further indicates that the excavation is due to increased intra-ocular pressure and that the position of the lamina cribrosa in glaucoma is not a constant one.

The depth of the glaucomatous excavation may be measured ac-

PLATE III.



The fundus of an eye with chronic glaucoma.

cording to the directions given on page 117. It is also recognized by employing the *parallax test* with the indirect method as follows: The optic nerve is found in the usual manner by the inverted image, and the object lens moved from side to side. The entire eye-ground apparently moves with the motions of the lens, and the bottom of the excavation also seems to move in the same direction, but at a much slower rate. The contrast in the rate of the two movements is in a direct ratio with the depth of the excavation.

The cup varies from one beginning to be pathologic to a fully formed excavation. In the latter instance the excavation is complete to the scleral margin, and its edges are abrupt; the vessels are crowded to the nasal side, bend sharply over the margin, and are lost to view behind the border of the cup, reappearing in fainter color at its bottom.

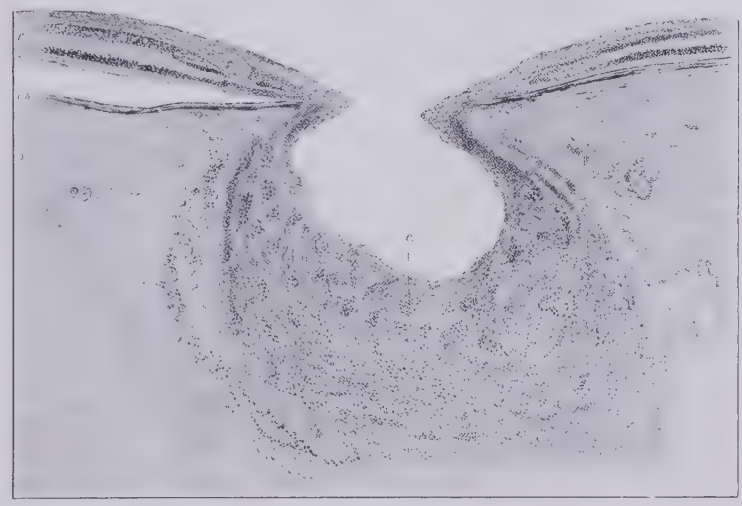


FIG. 189.—Section of optic nerve-head containing a deep glaucomatous excavation, the so-called kettle-shaped excavation: *r*, Retina; *ch*, choroid; *s*, sclera; *c*, cup, or excavation, pushing back lamina cribrosa.

The papilla is encircled by a yellowish ring due to atrophy of the surrounding choroid.

It is important to distinguish between a large physiologic cup, an excavation due to atrophy of the optic nerve, and a glaucomatous cup. A physiologic excavation is partial and formed in a normally tinted nerve-head; an atrophic excavation is complete, shallow, and formed in a nerve-head of abnormal whiteness, owing to its loss of capillarity; and a glaucomatous excavation is complete, deep, and often of greenish hue.

The descriptions thus far given apply to typical forms of each variety of excavation. Sometimes it is a matter of considerable difficulty to decide between them, especially between an atrophic and a glaucomatous excavation where the latter is shallow; or between a physiologic excavation and glaucoma, where the former is associated

with primary optic nerve atrophy. A diagnosis must then be based upon other symptoms, particularly an examination of the field of vision (page 418).

8. *Vessel Pulsation on the Surface of the Disk.*—(*a*) *The Veins.*—There is often marked venous pulse, especially at the dark knuckles of the veins as they bend over the margin of the excavation, but this is a common ophthalmoscopic appearance in healthy eyes (see page 109), and hence cannot be utilized as a diagnostic symptom. But Elliot's opinion is that when "other features of a case point toward glaucoma, the presence of marked venous pulsation furnishes contributory evidence."

(*b*) *The Arteries.*—Pulsation of the arteries is a rare appearance except in aortic regurgitation, and therefore may be regarded as an important indication of increased intra-ocular tension, in high degrees of



Fig. 190.—Photomicrograph of a glaucoma-cup; the optic nerve is atrophic.

which it is a striking symptom, the arterial trunks on the surface of the disk showing rapid alternate filling and collapse. It is usually, but not always, confined to the disk. (See also page 109.) This phenomenon is by no means always present in glaucoma, never in Elliot's experience except in the course of congestive attacks visible before the clouding of the cornea forbids ophthalmoscopic examination. Certainly in so-called subacute glaucoma it is not infrequent. The cause of spontaneous arterial pulsation resides in the resistance to the passage of the blood through the vessels, a resistance which, in turn, depends partly upon increased tension and partly upon spasmodic contraction of the vessels themselves. In most cases of glaucoma in which this pulse is not spontaneously visible it may be induced by slight pressure upon the globe.

In addition to the *objective* signs of glaucoma just described, certain *subjective* symptoms are more or less constantly present.

1. *Pain.*—In acute attacks the pain is a severe neuralgia of the trigeminal distribution, and often, in violent congestive cases, an intense agony associated with great depression, pallor of the countenance, nausea, and vomiting. In subacute attacks there is a less marked similarly located pain. In chronic cases there may be only a general feeling of discomfort, a sense of fulness, occasional shoots of neuralgia, or attacks described by the patient as "headache"; or pain is entirely absent.

2. *Alteration in the Sensibility of the Cornea.*—Anesthesia of the cornea varies from a slight depreciation in its sensitiveness to an entire loss of sensation, as complete as that produced by cocain. Sometimes the anesthesia is not uniform over the surface of the cornea, but exists in spots or segments. It is due to the edema of the structure, which presses upon the filaments of the corneal nerves.

3. *Alterations in the Light Sense.*—Although it is well known that the light sense is markedly affected in glaucoma, the interpretation of the results which have been obtained are by no means uniform. Elliot, who has designed a useful light-sense apparatus for clinical purposes, quotes with approval the observations of Beauvieux and Delorme thus: The differential light sense (*i. e.*, the smallest difference perceivable between two illuminated areas of different intensity) is early attacked, a lessened acuity being observable prior to the period when ophthalmoscopic evidence is definite or there is diminution of central or peripheral vision. The absolute light sense (*i. e.*, the perception of minimal stimulus or threshold) is lessened only after definite changes in the nerve-head are evident. Night-blindness, although uncommon, has been observed, but glaucoma patients are unduly sensitive to diminished illumination in the ordinary sense of this term. The subjects of simple glaucoma even in its earliest stages, although they may have normal visual acuteness as determined with the ordinary test types, if they are tested with Bjerrum's or de Wecker's photometric types almost always reveal a decided lessening of acuity of sight.

4. *Alterations in Central Visual Acuteness.*—This symptom varies considerably, and in chronic cases excellent sharpness of sight may be preserved for a long time. It is important to remember this, because it is not safe to depend upon central vision as a guide of the rate of progress of a chronic glaucoma. In each attack of subacute glaucoma the vision quickly fails, and gradually is recovered as the attack passes away. Each recurrence leaves a more permanent impression. In acute glaucoma a characteristic symptom is the sudden loss of vision, which in a few hours may be reduced to a light perception, and in certain malignant types rapidly becomes extinct.

5. *Alteration of the Refractive Power of the Eye and Diminution of the Amplitude of Accommodation.*—The former depends upon the change in the shape of the cornea, and the latter upon the effect of pressure upon the ciliary nerves. Alterations in the curvature of the cornea tend to produce an astigmatism "contrary to the rule" (see page 151), hence this is an important event in chronic glaucoma and in periods preceding

its development. Diminished power of accommodation is evidenced by the desire of patients to change their reading-glasses to such as are stronger than the degree of refractive error or age of life would warrant.

6. *Alteration in Peripheral Vision, or the Field of Vision.*—A careful map of the field of vision in glaucoma is indispensable, and the restric-

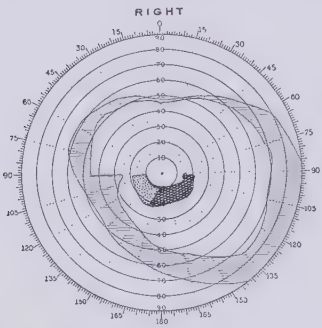


Fig. 191.—Chronic glaucoma, Bjerrum's scotoma and Rönne nasal step. (After Elliot, slightly modified.)

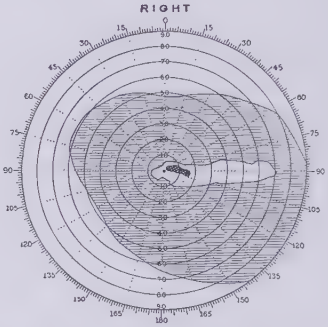


Fig. 192.—Chronic glaucoma. Narrow field on temporal side, scotoma passing from blind-spot (Bjerrum's sign). Vision 6/12; duration four years.

tions present themselves in several forms: (*a*) The most usual and typical variety is partial or complete loss of the nasal field or of the upper or lower quadrant of the nasal side; (*b*) concentric restriction of the entire field; (*c*) restriction so constituted that the remaining field assumes an oval or trowel shape; (*d*) sectional defects, often of the superonasal area;

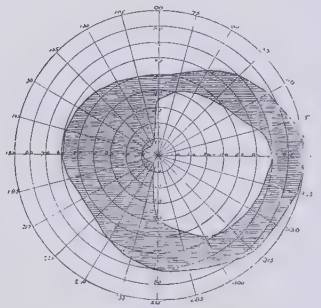


Fig. 193.—Field of vision of right eye in a case of subacute glaucoma. Loss of the nasal half and concentric restriction of the preserved field.

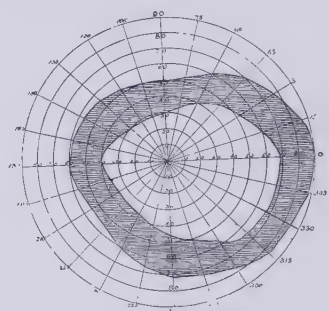


Fig. 194.—Field of vision of right eye in a case of chronic glaucoma, showing concentric restriction of the field.

(*e*) loss of the entire field except a patch on the temporal side; (*f*) the formation of scotomas, which may be central, paracentral, annular, or peripheral (Figs. 198–202); (*g*) special visual field defects—Bjerrum's scotoma, Seidel's sign, Elliot's sign, Rönne's step.

The contraction of the color-fields is usually proportionate to that of the form-field, but this rule meets with exceptions. Under the in-

fluence of operative measures or miotics very decided improvement in the extent of the visual field may take place.

The tendency of the visual field is to contract progressively as the disease advances, and finally all portions are obliterated except a small

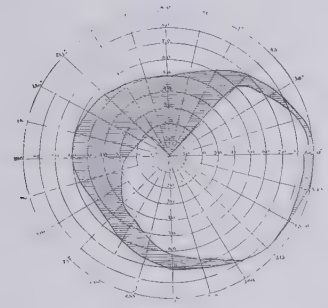


FIG. 195.—Field of vision in right eye in case of chronic glaucoma, showing sectional defect (superonasal quadrant).

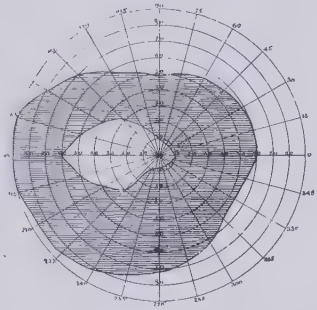


FIG. 196.—Field of vision of left eye in chronic glaucoma. Trowel-shaped patch preserved chiefly on the temporal side.

part upon the temporal side, which also disappears in the ultimate blindness (consult Fig. 197).

The contraction of the field of vision is an important index of the rate of progress in glaucoma, more important than depreciation of central vision; but it is not sufficient to trust to the periphery of the field

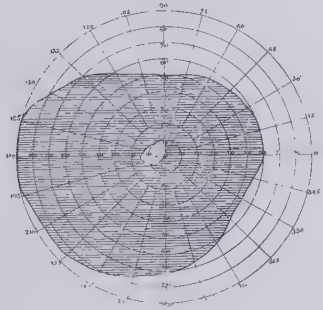


FIG. 197.—From the same case as Fig. 196, six months later; only a small patch of preserved field on the temporal side.

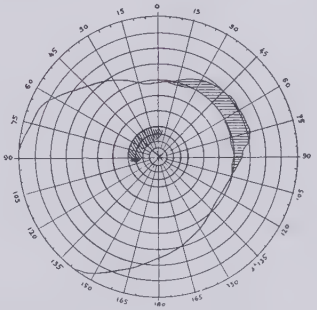


FIG. 198.—Just beginning contraction of nasal field; scotoma extending from blind-spot in a semicircular manner upward and inward (Bjerrum scotoma).

for information. A search for *scotomas* is imperative. They must be found either by the method suggested by Bjerrum or with the aid of a campimeter, and by means of "small object perimetry" (Elliot) and with the help of "scotometers." For the various methods of visual field examinations, consult pages 83–90.

Bjerrum's scotoma is topographically different from that which

occurs in simple optic-nerve atrophy, and may be utilized as a differential test between the two conditions, as Bjerrum has already suggested. The scotoma is peculiar, in that wherever situated it is in direct continuity with the blind-spot. The scotoma may extend from the blind-spot and pass in an arc above and below the fixation point to end at the nasal side of it, about the horizontal meridian, or the defective area may merge with the blind area commonly existing in the nasal part of the field. Should the arcuate scotoma extend until its ends unite, an *annular scotoma* is produced. As this scotoma (Bjerrum's scotoma) nearly always starts at the blind-spot, it must be due, as Bjerrum has pointed out, to a limited destruction of the nerve bundles of the papilla at the margins or sides of the excavation, that is, the superotemporal and inferotemporal bundles. It constitutes a most important sign of glaucoma, even if it is not a pathognomonic visual field defect. Naturally, should there be limited destruction of nerve-fibers in the papilla in other diseases, for example, retrobulbar neuritis, neuroretinitis, etc., they

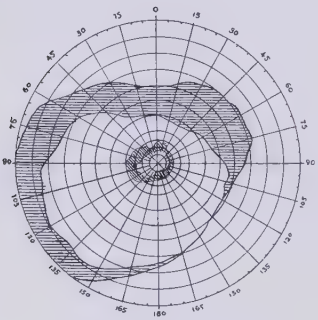


FIG. 199.—Annular scotoma in chronic glaucoma formed by the union of inferior and superior Bjerrum scotomas; moderate contraction of the peripheral field.

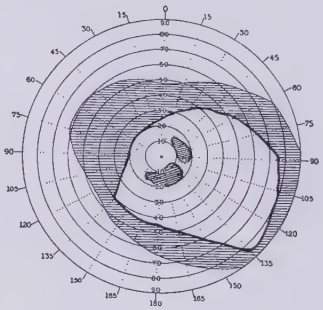


FIG. 200.—Field of vision of right eye in chronic glaucoma. A detached scotoma below fixation, separated by a bridge from a Bjerrum scotoma, passing upward and inward from blind-spot.

would be interpreted by closely similar scotomas. Bjerrum's scotoma may be partially or wholly relative, and although it usually arises from the blind-spot, it may also begin in another part of the arc (Van der Hoeve). Peter maintains that "most Bjerrum signs begin as detached areas above and below the fixation center." Defects occur in the intermediate zone of the visual field, due to interference with the more peripheral nerve bundles.

Rönne's Nasal Step.—As Rönne points out, a small paracentral arciform scotoma is a common phenomenon, and to a break in the nasal field, whereby an alteration takes place in the boundary line of the field at the inner side from vertical to horizontal along the horizontal meridian, which it pursues for a varying distance, before it again assumes a vertical or nearly vertical direction, he has given the name "nasal step." Quoting Elliot, the explanation of this "step" is as follows: If the arching optic nerve-fibers proceeding to the tem-

poral extremity of the retina are equally damaged, both at the upper and lower margin of the disk, the result will be an even impairment of the nasal side of the field; if either the upper or lower fibers are most damaged the field supplied by these fibers will show a corresponding restriction and such unequality of lesion is demonstrable in a careful chart of the visual field and constitutes Rönne's sign. Hence the step may be either above or below the horizontal meridian. It is an important sign and cannot be detected by ordinary perimetry with large test objects (Fig. 191).

Seidel's sign consists in a wing-shaped extension of the blind-spot upward or downward or in both directions. Such scotomas, it is said by Seidel, may be found in the field of an unaffected eye, its fellow being glaucomatous. As the glaucomatous process advances these scotomas may develop into scotomas of the Bjerrum type, and they, as

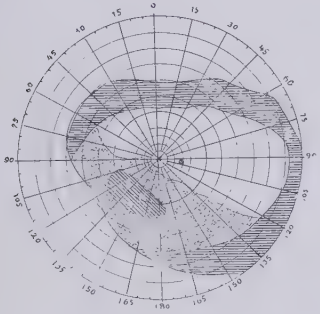


FIG. 201.—Visual field of right eye in chronic glaucoma, showing the mechanism of the loss of the lower and inner portion of the field, preceded by a scotoma, which gradually extends. Scotoma represented by parallel lines; area of dull vision which subsequently is completely lost, by dots.

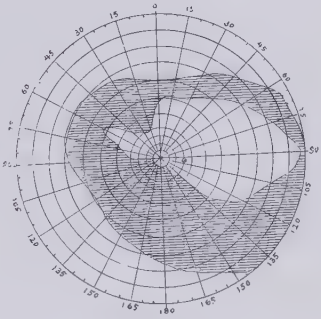


FIG. 202.—Later stage of Fig. 201. The scotoma has extended, and the area of the visual field in which sight was only dulled, and which is represented by dots in the preceding figure, has become completely dark.

has been explained, may be responsible for annular scotomas. Instead of a single somewhat pointed enlargement of the blind-spot (Seidel's sign), a number of points are demonstrable, according to Elliot, by means of his scotometer (*Elliot's sign*). Direct vision may be blotted out by a central scotoma, and peripheral scotomas may be the forerunners of subsequent defects with peripheral visual field (Figs. 201, 202). It is probable that some of the scotomas in glaucoma depend upon lesions in the retina due to alterations in its vascular supply.

6. *Iridescent Vision.*—This consists of a definite ring surrounding artificial lights, which thus become invested with a colored halo (*halo vision*). First there is a dark band, followed by a concentric zone of prismatic colors.

This phenomenon is due to an edema of the cornea (or alteration of corneal epithelium) which depends upon increased intra-ocular pressure.

Patients can usually distinguish blue, red and yellow or yellowish-red, and often green.[1]

Subjective sensations of light are experienced at times by totally blind glaucomatous patients. The explanation is probably a mechanical one, and the sensation depends upon a dragging on the retina. In one case noted by the writer, both eyes being blind from glaucoma, the patient declared "all things seemed to be a sea of red fire."

The clinical varieties of glaucoma may now be described.

1. **Acute Glaucoma** (*Acute Congestive Glaucoma*).—This type of the disease is usually divided into two stages:

(*a*) **Prodromal Stage.**—This period, really an early stage of congestive glaucoma, is characterized by sudden failure in the amplitude of accommodation and a desire to resort to stronger reading-glasses; temporary obscurations of vision, either dim vision or complete loss of sight, lasting for many minutes; attacks of foggy vision, due to increased intra-ocular tension, all things apparently being invested with a haze; and the phenomena of colored halos around artificial lights. There may be some periorbital pain, the pupil is slightly dilated, and the cornea and the aqueous humor faintly turbid. The appearance of the optic nerve at this stage is not characteristic.

These prodromes bear some relation to emotional excitement and insomnia, and may occur when the head is congested or after a full meal. After the eye regains its natural state, in a week or two the symptoms may reappear, again to subside and to be replaced by a fresh exacerbation or a true "glaucomatous attack." The early period of glaucoma may last one or more years.

(*b*) **Period of Attack, or the "Glaucomatous Attack."**—This often begins during the latter part of the night, but by no means necessarily so, sometimes preceded by prodromes, but sometimes without previous warning, and is characterized by violent pain in the head, so severe that it may induce nausea and vomiting. The face may be pallid, the extremities cold, or there may be flushed features and general fever. The eyelids are swollen, the conjunctiva injected and sometimes chemotic, the cornea steamy and anesthetic, the pupil semidilated and motionless, the aqueous turbid, and the iris discolored. The ocular tension rises, the tonometer registering 80 mm. of Hg. and higher, and

[1] Halo vision occurs in mild attacks of iritis with slight deterioration of vision. It may also be caused by a layer of mucus overspreading the cornea during chronic conjunctivitis. According to Myles Standish, the halo due to mucus has only the outer or red and yellow bands. The presence of blue in the halo may, therefore, he thinks, be regarded as indicating increased intra-ocular tension. The corona of light around a flame, the colored ring discernible when looking through a "fogged" spectacle lens, or other piece of glass, even the scintillating scotoma of migraine (Morax), have been mistaken for glaucomatous halos. The normal eye under appropriate conditions sees diffraction halos around bright lights, but they are very difficult to find, and are much less bright than the halo of glaucoma (Emsley and Fincham). If a source of bright light is examined through a piece of glass on which lycopodium has been dusted, a halo similar to the one caused by glaucoma is produced (Elliot). The subject of halo vision in all its aspects has been thoroughly studied by Drualt, Morax, Elliot, and Emsley. (See also page 421.)

vision is rapidly lost, often only light perception remaining, and even this may be abolished. Sometimes the attack is bilateral, or only a few hours elapse before the second eye is affected. Again, the interval between the two attacks may be prolonged weeks, months, or even years.

Gradually the symptoms pass away, with the exception of slight impairment in the mobility of the iris, some limitation of the field, and a little rise in tension. Blindness almost never occurs in the first onset. At this time characteristic ophthalmoscopic appearances are not present. After a number of attacks, examination of the eye-ground during a remission (the fundus is not visible during an attack) may reveal the usual changes.

If the disease is unchecked, the eye passes into a *glaucomatous state*, with fixed and dilated pupil, discolored iris, greenish reflex from the lens, vitreous opacities, shallow anterior chamber, and hazy cornea. Vision is now gradually destroyed and the eye reaches the *state of absolute glaucoma;* the ball is stony hard, the iris atrophic, the lens cataractous and pushed forward, the anterior chamber obliterated, the sclera discolored, the episcleral vessels coarsely injected, the cornea opaque, or perhaps ulcerated. Finally, there is disorganization of all the structures of the eyeball, and the sclera gives way with the formation of staphylomas, or the eyeball slowly atrophies as the result of choroiditis, change in the vitreous, and detachment of the retina. Unchecked acute glaucoma, instead of pursuing this course, occasionally passes into a chronic congestive type.[1]

Spontaneous rupture of a glaucomatous eyeball occasionally occurs; at the same time there may be choroidal hemorrhage.

Glaucoma fulminans is the name applied to an aggravated, rare form of the acute disease, in which the symptoms may be fully developed in a few hours without a prodromal stage. There is no remission, and the destruction of vision is swift and permanent.

2. **Subacute or Chronic Congestive Glaucoma.**—Naturally, congestive glaucoma is not always characterized by the very acute symptoms which have been recorded in a previous paragraph. The signs of the disease under certain conditions do not change, save only that their manifestations are more moderate. In such circumstances the title *subacute glaucoma* is often utilized, and if the exacerbations with the intervals of remission last for a long time the name *chronic congestive glaucoma* has been given. While there is no objection to the employment of these terms in descriptions of the disease, they should not be used as if they indicated special and separate varieties of glaucoma.

3. **Chronic Glaucoma or Non-congestive Glaucoma** (*Usually Known as Simple Chronic Glaucoma or Glaucoma Simplex*).—This type

[1] Elliot objects, with justice, to the terms "prodromal stage" and "absolute glaucoma," but they are in such common use that the author has continued them in the text. Elliot's classification is: early glaucoma, established glaucoma, late glaucoma.

of the disease is characterized by an absence of the signs of glaucoma in the anterior aspect of the eye, at least on ordinary inspection. There is no pain except as previously stated (page 417). The tension of the eyeball is always increased at some period of the disease; it may be of minor degree, is not constantly present, or it may be present at one portion of the day and not at another, or during the night and not in the daytime. The necessity of frequent tonometric tests at different times during twenty-four-hour periods is evident. The depth of the anterior chamber is not materially altered. By careful examination (loupe or corneal microscope) slight steaminess of the cornea may sometimes be detected with a little lack of transparency in the aqueous humor. So, too, there may be some undue tortuosity of the perforating branches of the episcleral plexus. If nebulous vision, halos, etc., or, in other words, irritative phenomena develop, the case ceases to be one of simple glaucoma, and should be grouped with the congestive types.

Usually both eyes are affected, simultaneously or successively; but it is difficult to fix the exact date of the onset of this variety of glaucoma because of the absence of the pronounced early symptoms which precede the other types of this disease.

If both eyes are implicated, the process is usually more advanced in one than in the other, and the pupil is generally slightly larger on the side of the greater disease. In the later stages a greenish sheen from the pupil is often distinct. The central vision may be good, and in the earlier stages of the disease, after the correction of any refractive error, may reach the normal standard, and this may be true even where the disease has lasted a long period of time and marked contraction of the field is present.

The media are practically clear, and the disease is detected with the ophthalmoscope by observing the characteristic cup in the nerve-head, the halo surrounding it, and sometimes the ready development of an arterial pulse by slight pressure. The field of vision gives important information, and it assumes one or other of the characteristics described on page 418 (see Figs. 191–202). The central color perception is good, and the contraction of the peripheral color perception usually corresponds with that of the field for white.

Simple chronic glaucoma may be transformed into subacute or acute glaucoma, but may continue throughout its course without aggressive symptoms.

Optic-nerve atrophy with excavation, or so-called *glaucoma simplex without rise of tension* should be distinguished from chronic non-congestive glaucoma because the tension does not rise above the normal limits (20–28 mm. [Morax]), the visual field phenomena differ from those of true glaucoma (pages 418–421) and there is no favorable response to miotics or operation. Morax believes the process is due to a special affection, localized in the blood-vessels or optic fibers, whose characteristics are a slowly developing double atrophy with excavation. H. S. Gradle is satisfied, as the result of his observations, that this condition

represents a disease *sui generis* of unknown origin, possibly due to a systemic selective toxin which attacks the optic nerves.

Causes.—(*a*) *Predisposing Causes.*—Primary glaucoma is rare before the fortieth year; not 1 per cent., according to Priestley Smith, begins earlier than the twentieth year.[1] Although rarely, glaucoma may occur in children and one form is encountered between the ages of fifteen and twenty, the dominant refraction being myopic, which differs somewhat in its development from buphthalmos (page 442) and is also called *juvenile glaucoma.* There may be a *hereditary tendency* to glaucoma, and, according to Nettleship, if the disease appears in more than one generation, it develops at an earlier age than in the preceding generation ("anticipation"). Lawford's research shows that *familial glaucoma* is continuous in descent and is transmitted by both sexes. Jews, Egyptians, and Brazilian negroes are peculiarly liable to glaucoma. The glaucomatous eye is usually hyperopic, although Priestley Smith's statistics do not indicate a striking preponderance of this refractive state. Myopia confers no immunity from glaucoma. There is a relation between smallness of the cornea and glaucoma. The average horizontal diameter of the normal cornea is 11.6 mm.; of the glaucomatous cornea, 11.1 mm. (P. Smith). A large lens is a predisposing factor, and small eyes, in which the lens may be disproportionately large, are more liable to the disease than normal globes.

(*b*) *Exciting Causes.*—Glaucoma may be excited in eyes predisposed to the disease by worry, insomnia, bronchitis, cardiac disease, syphilis, gout, influenza, angioneurotic edema, and neuralgia of the fifth nerve.

In general terms it may be said glaucoma rarely occurs in persons in perfect health, as examinations according to modern methods readily demonstrate. A. Knapp classifies cases of primary glaucoma into those which are "circulatory" and those which are "nervous." The first group includes cases which are characterized by congestive attacks and by retinal, vascular, and local conditions which favor increased tension. The second group includes cases characterized by dysglandular disturbances which affect any type of eye.

Sometimes glaucoma follows injury and hemorrhage into the uveal tract, and *traumatic glaucoma* may be caused by a contusion of the globe without rupture of its coats.

It has been stated that at corresponding ages usually there is higher average blood-pressure in glaucomatous subjects than in non-glaucomatous subjects, and that arteriosclerosis and, therefore, increased blood-pressure are exciting causes of glaucoma. This contention is disputed by many observers. It is generally conceded that high blood-pressure is not a factor in the causation of glaucoma, although the subjects of this disease are often also the subjects of arteriosclerosis. Dis-

[1] Priestley Smith's statement is as follows: The frequency of glaucoma increases "slowly at first, more rapidly later in each decade, until about the sixtieth year. Between sixty and seventy it is about as frequent as between fifty and sixty. After seventy its frequency diminishes." These results have been confirmed by other more recent observers.

turbances in the organs of internal secretion have been suggested by E. von Hippel as a possible cause of glaucoma.

Overuse of ametropic or improperly corrected eyes, by causing uveal congestion, may excite glaucoma in an eye predisposed to the disorder. On the other hand, Grönholm, believing that the filtration channels are widened in the act of accommodation and by contraction of the pupil, suggested that glaucoma patients should be advised to stay as much as possible in a bright light, and to read so long as they have accommodation power and their pupils contract to light. Arthur Thomson recommends that in glaucomatous subjects the action of the ciliary muscle shall be restored by suitable exercises (quoted from Elliot).

In a number of instances instillation of mydriatics has caused glaucoma. Acute glaucoma appears to be more frequent in winter than at other seasons of the year (Geisler).

Should a patient between his fiftieth and sixtieth year desire to change his reading glass frequently, or to use one stronger than is suited to his age or the condition of the refraction of his eye, there is reason to apprehend the onset of glaucoma. On the whole, the disease is more common in women than in men (Priestley Smith[1]). Those symptoms which have been described as prodromes are distinctive in themselves, and acquire an importance greater than any probable predisposition.

Pathogenesis and Pathology.—Three kinds of fluid are recognized within the eyeball: the blood within the blood-vessels, the lymph within the perivascular lymph-channels and the spaces in the uveal tract, and the intra-ocular fluid which is concerned with the nourishment of the vitreous and lens, supplies the aqueous chamber, and which, as was proved by Leber, proceeds from the epithelium of the ciliary body. It is probable that the lymph formation of the eye is produced in the ciliary body by a process of transudation, and not, as has been taught, by a process of secretion.

The chief stream of the intra-ocular fluid thus derived proceeds over the lens and through the pupil into the anterior chamber, traverses the latter to reach the angle formed by the junction of the iris and cornea, passes through the meshes of the ligamentum pectinatum, and by diffusion and filtration is taken up by Schlemm's canal. From this canal the greater quantity of the fluid passes into the anterior ciliary veins, a part of it being absorbed and eliminated by the iris (Nuel, Benoit). Arthur Thomson concludes from his researches that escape of fluid from the anterior chamber into Schlemm's canal and from there into the veins is the result of a "pumping action" whereby the muscles of the ciliary body and of the iris draw back the scleral process and so open the spaces in the pectinate ligament. Only a very small portion of the fluid flows backward through the vitreous and escapes by way of the perivascular lymph-channels in the optic nerve. According to Priest-

[1] Elliot states that "chronic non-congestive cases are found perhaps a little in excess in males as compared with females," but that congestive forms are much more frequently encountered in women than in men.

ley Smith, it is doubtful if there is any continuous stream from the vitreous into the aqueous chamber, but the anterior hyaloid membrane and suspensory ligament are easily permeated by it, and in health any excess of fluid in the vitreous chamber escapes by the filtration angle in the manner already described. The pressure of the fluid regulates the outflow, so that when the afflux is increased a compensating increase of the efflux occurs.

The fluids of the aqueous and vitreous chambers are nearly identical in composition and contain about 95 per cent. of water, 1 per cent. of extractives and salts, and a minute quantity of albumin. The intra-

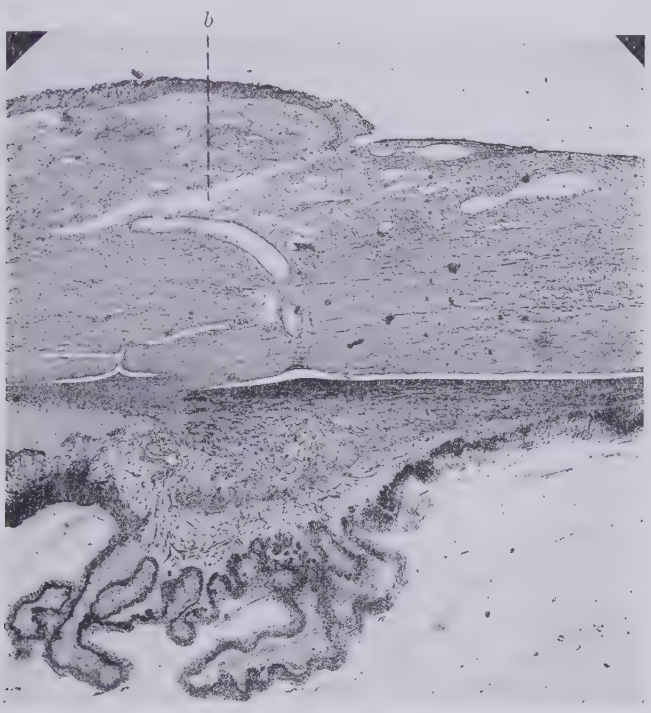


FIG. 203.—Photomicrograph of a specimen prepared by Dr. Brown Pusey. Communication between Schlemm's canal (*a*) and scleral vein (*b*).

ocular pressure, which is equivalent to that of a column of mercury 25 mm. in height, is the same in the vitreous and the aqueous chambers, and preserves the shape and tension of the eyeball.[1] If anything occurs to disturb its regulation, to quote Priestley Smith, "the pressure in the ocular chambers rises above the physiologic limits and we have the complex disturbance of function and structure called glaucoma."

What exactly are the factors potent in disturbing the regulation of

[1] Elliot considers it important to point out that the pressure of the aqueous and vitreous chambers is not exactly the same throughout. There are, according to him, "distinct though slight differences of pressure at various points in the mass of fluid within the eye."

pressure has never been entirely determined, and numerous theories have been advanced.

The theory which has been and is still widely maintained is that one which assumes a diminution in the outflow and, therefore, a retention of fluid (*retention theory*). It obtained proper recognition when Knies and Weber demonstrated that in glaucomatous eyes, with shallow anterior chambers, there is an adhesion of the iris base to the periphery of the cornea, which prevents filtration at the angle of the anterior chamber and causes retention of the intra-ocular fluid (Figs. 190 and 191). This adhesion Knies regarded as an inflammatory process—that is, as a species of anterior iridocyclitis, while Weber considered it to be secondary to the pressure induced by an abnormally swollen

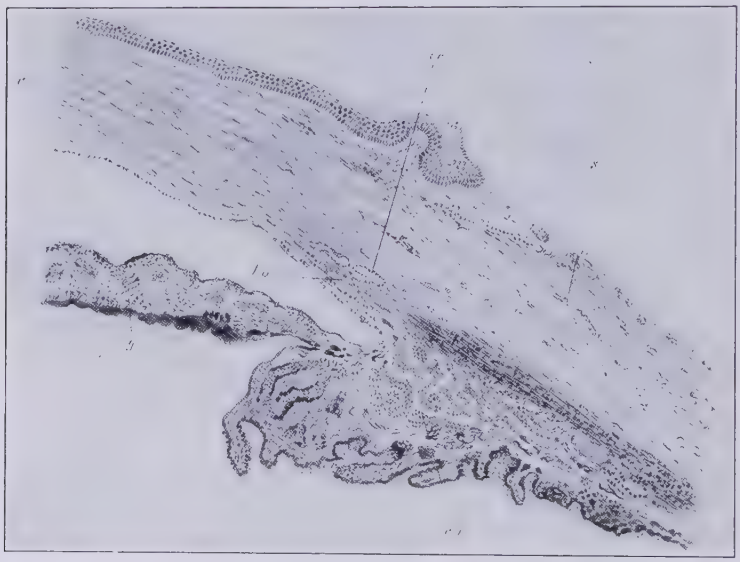


FIG. 204.—Angle of the interior chamber of a normal eye: *c*, Cornea; *s*, sclera; *g*, iris; *c.b*, ciliary body; *l.p*, ligamentum pectinatum; *s.c*, Schlemm's canal.

ciliary body. The fact that a mydriatic does harm to an eye predisposed to glaucoma by dilating the pupil, rolling back the iris, and partly closing the filtration angle, and that eserin does good by contracting the pupil and drawing away the iris from this angle, indicates that the explanation of glaucoma is to be found not in an increase of secretion, but in a disturbance of excretion.

Mydriatic glaucoma has also been ascribed to narrowing or closure of the iris crypts, an explanation which Priestley Smith declines to accept, for if it were correct, mydriasis should always be followed by rise of tension.

According to Priestley Smith, obstruction of the circumlental space —*i. e.*, the space between the margin of the lens and the surrounding

structures—and consequent rise of pressure may follow increased size of the lens due to advancing years, unusual smallness of the ciliary area in hyperopia, or abnormal enlargement of the ciliary processes, and vascular disturbance which congests the uveal tract. It is possible that hypersecretion is sometimes concerned in the onset of glaucoma, and that serosity of the fluids plays a rôle in those forms which present a deep anterior chamber and wide filtration angle; but obstruction at this angle is part of the glaucomatous attack in the vast majority of cases.

C. Hess pointed out that bulbous outgrowths may develop on the summits of the ciliary processes in the course of life. He observed swelling of these processes, followed by narrowing of the circumlental space in two eyes with severe primary glaucoma with the aid of a specially

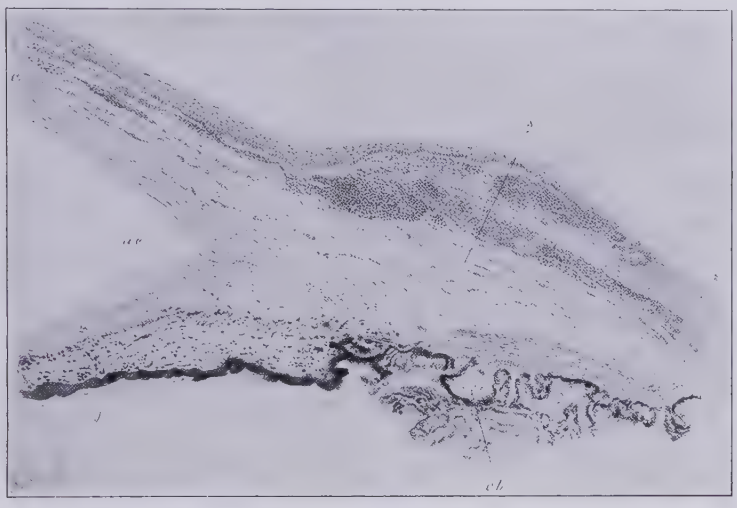


Fig. 205.—Angle of the anterior chamber in long-standing absolute glaucoma: *c*, Cornea; *s*, sclera; *j*, iris; *c.b*, ciliary body; *a.c*, angle of chamber closed by adhesive inflammation of the iris base to periphery of cornea, obliterating filtration area.

constructed lamp. Individual differences of the ciliary body are as important as differences in the size of the lens, and may have a distinct bearing on closure of the angle of the anterior chamber and the production of glaucoma.

Laqueur and other observers have maintained that glaucoma depends upon obstruction of the intra-ocular lymphatics, which find their way out with the venæ vorticosæ, owing to rigidity of the sclerotic coat. Brailey described a chronic inflammation of the ciliary processes and iris periphery, with distention of the vessels, as the earliest lesion in glaucoma. Stilling taught that a hardening of the sclera surrounding the papilla, through which waste fluid escapes, is responsible for glaucoma, and Strokousky attributes glaucoma to an *indurative sclerītis*.

Evidently all retention theories assume that the cause of glaucoma depends upon an obstruction to the outflow of liquids from the eye occasioned by an interference with their escape through Fontana's spaces at the filtration angle, or through the perivascular lymph-channels in the posterior part of the eye, or through both of these exits; hence the fluid accumulates, the intra-ocular tension rises, and glaucoma results. In other words, upon the increased tension depend all the disturbances in the eye in this disease.

But this explanation is not satisfactory to many observers, and certain objections have been advanced. For example, it is maintained that although adhesion of the root of the iris and blocking of Fontana's spaces are usually present in congestive glaucoma, it does not follow that this condition is the cause of the glaucomatous process; it may as well be, indeed, it is more likely to be, a result of it. Moreover, while it is practically always present in the eyes with long-standing glaucoma, it may be absent in an early stage of the disease. But especially it is true that this theory does not satisfactorily explain the mechanism of so-called simple glaucoma, in which increased tension does not necessarily play a conspicuous rôle.

Wahlfors believed that the search for the cause of simple glaucoma must be made in the choroid, and maintained that the primary lesion is an atrophic process in the choriocapillaris, leading to nutritional disturbances in the layer of the rods and cones, whereby the important symptoms of this variety of the disease, diminution of light-sense, defects in the field of vision, and excavation of the nerve-head (see page 413) can be explained. Glaucomatous increase of tension he ascribed to paralysis of the muscular network of the choroid whereby a slowing of the intra-ocular liquids is caused, the retarded flow permitting the deposition of formed elements in the channels of exit, and, therefore, retention of the liquid; finally, the venæ vorticosæ are compressed by reason of the increased tension, and venous stasis is the result. According to the manner and activity with which these factors influence the eye, he believed the various types of glaucoma are produced.

Knies and other writers, unable to reconcile any of the theories of glaucoma with the so-called simple variety of the disease, inclined to separate it from the glaucoma class and place it among diseases of the optic nerve. In the present state of our knowledge, and keeping clearly in view the presence of increased tension, this does not seem advisable. Indeed, as Priestley Smith forcefully puts it, the nature of chronic and acute glaucoma is not essentially different, in that there is a close, though hidden, resemblance. "In both we find the predilection for small eyes; in both there is obstructive displacement of the iris—slowly established in the one case, rapidly in the other." Some authors explain this form of glaucoma by assuming a neuritis which blocks the lymph-channels in the optic nerve and its sheath, and which prevents the removal of effete matters which normally, to slight degree, occurs through these pathways, and thus causes increased tension and excava-

tion. Hence the disease is sometimes called *posterior glaucoma*, to distinguish it from the other variety, *anterior glaucoma*.[1]

Priestley Smith believes that "typical chronic glaucoma depends on slowly increasing contact of the iris with the cribriform ligament, arising through enlargement or advance of the lens, and involving in its early stages no serious compression of the iris."

W. Zimmerman considers that the primary cause of glaucoma depends upon a difference between the general blood-pressure and that of the eye. Parsons' experiments indicate that intra-ocular tension may *passively* respond to variations in the general blood-pressure, probably due to alterations in the volume of the intra-ocular blood-vessels, but it is uncertain that such passive changes are sufficient to account for glaucomatous attacks when these are apparently produced by excitement or emotion (see page 425).

The notable increase of the amount of albumin which Uribe Troncoso has found in the aqueous humor of glaucomatous eyes induces him to advance the theory that the symptoms which characterize glaucoma are best explained by its presence. The lesions in the blood-vessels which are found in glaucoma permit the passage of the albumin from the blood, and pathologic variations in the vitreous have also an important bearing on the glaucomatous process. Leber, however, who reviewed Troncoso's work, was unable to persuade himself that this author's views are correct. A. Knapp found in an eye with primary glaucoma an albuminous exudation which, by obliterating the anterior chamber through distention of the posterior chamber, produced the increased intra-ocular tension.

Brown Pusey, experimenting with the increase and decrease of intra-ocular tension which may be induced by varying osmotic pressures, believes that on them depends the explanation of the primary cause of glaucoma. M. H. Fischer holds that glaucoma is due to an edema of the eyeball, the amount of water contained in the hydrophylic colloids (chiefly the proteins) being increased by an augmentation of the quantity of acid present in the organ. The ordinary exciting causes of glaucoma are responsible for this abnormal production or accumulation of acid.

[1] Based on the assumption that the obstruction to the outflow of lymph from the globe may be more marked either in the anterior or posterior lymph system, W. R. Parker has classified glaucoma simplex clinically—as *simple anterior glaucoma* and *simple posterior glaucoma*. If the anterior spaces are blocked and the posterior spaces patulous the lymph flow will be backward and the anterior chamber may remain normal in depth or be but slightly shallow. On the other hand, if the posterior spaces are blocked and the flow of lymph is forward the lens and iris will follow the flow and the anterior chamber will be shallow. If the anterior spaces are free an iridectomy will be of no avail, while if they are blocked a properly performed iridectomy may restore a sufficient opening to relieve the hypertension. The clinical differentiation between the two forms of glaucoma simplex suggested is made by observing the depth of the anterior chamber. In cases of simple posterior glaucoma an iridectomy is performed, while in the cases of simple anterior glaucoma a trephine or other operation which has for its object the establishment of a permanent cicatrix is indicated. See American Journal of Ophthalmology, Vol. I, No. 9, 1918.

According to Thomas Henderson, the underlying predisposing and causal factor of glaucoma resides in a primary obstruction and closure of the pectinate ligament, or, as he prefers to call it, the *cribriform ligament*. This occlusion is the result of a sclerosis of the fibrous structure composing the filtration area which results, first, in a diminution, and, finally, in a complete obstruction of the outflow through it, leaving the iris, with its crypts, as the only efferent channel for the lymph-streams. While in his opinion this sclerosis is the fundamental cause in all cases of glaucoma, he admits a second and variable agent, vasomotor in nature, which determines the acute attacks of increased intra-ocular tension. Henderson's theory does not seem to Priestley Smith to be sufficient.

Levinsohn, finding a striking deposit of pigment cells, derived from the cells lining the ciliary processes, in an eye with absolute glaucoma, suggests that this may be an important factor in the production of acute glaucoma.

According to Koeppe, in glaucomatous eyes by means of the Gullstrand diaphragm-lamp the migration of pigment derived from the pigmented epithelium is demonstrable. This pigment is found at the posterior surface of the cornea, as fine dust-like deposits in the iris stroma, visible on the anterior limiting layer, and in the aqueous humor. Such particles of pigment it is assumed may block the angle of the anterior chamber. These pigment granules Koeppe has found in very early (so-called preglaucomatous) stages of the disease and, therefore, according to him have an important diagnostic significance (*Koeppe's sign*). In many eyes, all, however, with evident glaucoma, investigated by the author with the slit-lamp the pigment changes described by Koeppe have been present, sometimes only in delicate representation. H. Gradle records similar observations. Whether they should be accorded the importance which Koeppe has given them as an early diagnostic feature depends upon further investigation. Küschel believes that loss or disturbance of the elasticity of the supporting tissues of the eyeball is the cause of the various types of primary glaucoma. In all senile eyes this produces the "glaucomatous disposition."

Evidently all cases of glaucoma cannot be explained by any one theory, and the various clinical manifestations of the disease, as well as the results of treatment, indicate that sometimes one factor and sometimes another is the more potent in its activities. Of those which have been described, obstruction of the circumlental space dependent upon increasing size of the lens due to advancing years, obstructive displacement of the iris slowly or rapidly established (Priestley Smith), checking the outflow of fluid from the interior, swelling of the ciliary processes closing the filtration angle, alteration in the composition of the intra-ocular fluid or its increase in abnormal conditions of the vascular system, blocking of the efferent channels by edema and exudation, sclerosis or pigment deposition, and vascular or vasomotor changes furnish the most satisfactory explanations of the various phases of glaucoma.

The *pathogenesis of secondary glaucoma* is easily understood, as the conditions which give rise to it (see page 44) readily obstruct the outflow of the intra-ocular fluid and occasion its retention. Moreover, in some varieties of secondary glaucoma (uveitis), in addition to the accumulation of inflammatory cells in Fontana's spaces, there is an excess of secretion, highly charged with albumin, from the inflamed ciliary body. In brief, secondary glaucoma is caused either by obstruction of the filtration angle or by alteration of the constitution of the intra-ocular fluid.

As already pointed out, the apposition of the periphery of the iris to the cornea in *primary glaucoma* is at first unassociated with inflammation; but if the apposition is long continued, proliferation of the endothelium of Descemet's membrane and the iris takes place and these two layers become adherent. Later the endothelium in large measure disappears, there is a round-celled infiltration of the deeper corneal layers, and around Schlemm's canal the tissue cells proliferate, and the iris becomes firmly bound down at its new position at the corneoscleral junction. In the early stages of acute primary glaucoma the ciliary body and processes are engorged and swollen; later, and in long-standing cases, atrophy and shrinking occur. The changes in the choroid and their relation to the pathogenesis of the disease and the development of the excavation have been described. While there are no characteristic changes, as a rule, in the retina, in advanced cases atrophy of its elements are visible, and endo- and perivascular changes are evident, which may cause hemorrhage. As already noted, edematous swelling and sometimes actual neuritis precede cupping of the nerve-head. Later there is backward depression of the lamina cribrosa and atrophy of the optic nerve-fibers. Alterations in the intrascleral passage of the venæ vorticosæ are sometimes discoverable, which depend upon proliferation of the endothelium in this position.

Diagnosis.—It is of the utmost importance that glaucoma shall be recognized, if possible, in its very incipiency. The most usual prodromal (early) symptoms are a frequent desire to change the reading glasses, periods of obscuration of vision, photopsies, and the halos surrounding the lamp lights. (See also page 422.)

The glaucomatous attack itself has frequently been mistaken for a "cold in the eye," for iritis—and the disease has been aggravated by the instillation of atropin or other mydriatic, which in almost all circumstances is contraindicated—for neuralgia, and for reflex ocular pain. The condition of the pupil, the diminished depth of the anterior chamber, and the increased tension of the globe are the symptoms which should prevent so fatal an error. As pointed out by Parisotti and Trousseau, *ophthalmic migraine* sometimes simulates glaucoma, inasmuch as it may be associated with increased intra-ocular tension, arterial pulsation in the fundus, and contraction of the visual field.

The differential diagnosis of simple chronic glaucoma and atrophy of the optic nerve has been referred to and presents considerable diffi-

culty. The absence of constant increased tension in the simple form of the disease, or at least its doubtful presence, removes an important diagnostic point. Examination with the tonometer is of great importance in these circumstances. The greatest help is obtained by investigating the visual fields in the most painstaking manner. In glaucoma the color-fields present a restriction corresponding with that of the white fields, while in atrophy the peripheral color vision, especially for red and green, is markedly deficient. The diagnostic value of the shape of the field, and especially of the scotomas, notably *Bjerrum's scotoma*, has been described (see page 419).

Examination of the light-sense is important. In glaucoma the "light-minimum" is said by some observers to be deficient, but the "light-difference" not far from normal; in pure optic-nerve atrophy there is imperfect ability to distinguish between different intensities of illumination ("light-difference"). (Compare with page 70 and see also page 417.) In other words, according to Samelsohn, the light-perception power in glaucoma is much lessened, while the light-difference power is relatively not greatly interfered with; in optic-nerve atrophy the reverse is usually the case. In all cases of early glaucoma examined by A. S. Percival he always found a light-minimum defect, and it was afterward followed by a light-difference defect. Wahlfors, confirming observations made long ago by Mauthner and Förster, insists that reduction of the light-sense is one of the most frequent symptoms of simple glaucoma, and that night-blindness may first call the patient's attention to his eyes. Moreover, this reduced light-sense may exist for years before the real nature is evident. A careful clinical history of patients who are the subjects of glaucoma often confirms this statement.

It is an inexcusable error to confound the failing vision of chronic glaucoma with that of cataract, the greenish reflex of the lens, which may be seen in the pupillary space, being mistaken for an opacity of the lens. Eyes have been permitted to pass into blindness, and their possessors deluded with the hope that they were waiting for the ripening of a cataract which never existed. An early ophthalmoscopic examination would settle the diagnosis at once.

Prognosis.—Glaucoma does not tend to spontaneous cure, but, if unchecked, to blindness; hence the prognosis is unfavorable if proper treatment cannot be applied. Prognosis also depends upon the type of the disease and the stage of its development. Other things being equal, uncomplicated acute cases furnish the most reasonable hope of complete cure, and if a technically correct operation can be performed *early*, the result is usually satisfactory. In chronic cases much depends upon the amount of degenerative change in the tissues, and the prognosis must be guided by the state of vision, the extent of the field, and the condition of the iris. The earlier treatment (operation or otherwise) can be begun in this as well as the acute types the better will be the result. The effect of treatment upon the progress of glaucoma is included in the following section:

Treatment.—In the majority of cases of acute glaucoma an operation is needed to check the disease.

It may happen, however, that an operation is not at once possible or advisable, and hence miotics should be quickly and thoroughly used. In the early stage eserin salicylate or sulphate should be employed and will usually relieve the symptoms. In acute cases eserin, in a strength of from 4 to 5 grains (0.26–0.324 gm.) to the ounce (30 c.c.), acts favorably, and often with surprising rapidity, provided the pupil responds to its influence. Pilocarpin hydrochlorid, 2 to 5 grains (0.13–0.324 gm.) to the ounce (30 c.c.), may be substituted. An excellent plan is to combine the activities of both these miotics. Miotics act by drawing the iris away from the filtration angle, and, by contracting the pupil, cause widening of the spaces of Fontana and absorption of the fluid; also iris surface filtration is increased (see also page 426). A drop or two of the selected solution should be instilled every ten or fifteen minutes for an hour and then the frequency of the instillations lessened until relief is obtained; if this does not occur promptly, iridectomy or one of its substitutes should be performed. Arecolin in 0.5 per cent. solution has also been used; with this drug the author has had no experience. Dionin and adrenalin chlorid have been much employed in the treatment of acute glaucoma. The former (in 5 per cent. solution) often acts efficiently as a lymphagogue and analgesic; the latter (1 : 1000) must be used with caution, as occasionally it increases the intra-ocular tension. It may be added to the solution containing the miotic.

In addition to the use of eserin or pilocarpin during an acute attack the temple may be leeched, warm fomentations applied, and rest and relief from pain secured by the exhibition of morphin and chloral, the latter drug having some influence in reducing tension. Full doses of salicylate of sodium, however, act more favorably than any other constitutional remedy (Sutphen, Friedenwald); indeed, they are most useful in any form of glaucoma associated with pain. An interesting observation of Morax is that reduction of tension in glaucomatous eyes in syphilitic subjects has followed the injection of salvarsan, an observation which the author can confirm. To lower intra-ocular tension Fischer and Thomas recommend subconjunctival injections of *sodium citrate* (4.05–5.41 per cent. solution). Of these solutions, 5 to 15 minims (0.30–0.92 c.c.) are injected, and if the injections are frequently employed, the weaker of the two, diluted with 2 to 4 parts of physiologic salt, is employed. There is a good deal of difference of opinion as to the value of these injections in such circumstances; the author has had no experience with them for this purpose. Cocainization of the region of the *nasal* (sphenopalatine, Meckel's) *ganglion*, according to the method of Sluder, gives relief in severe headache of obscure origin; it also reduces pain in glaucoma and produces a temporary reduction of increased intra-ocular pressure.

In chronic inflammatory (subacute) glaucoma eserin (the sulphate or salicylate) or pilocarpin should be employed until it is decided what

operation shall be done and when it shall be performed The other remedies advised in the preceding paragraphs are also useful and should be employed.

There is much difference of opinion in regard to the value of miotics in the treatment of chronic, non-congestive glaucoma. According to some surgeons, who deprecate operation in this form of glaucoma, they represent the chief therapeutic measure, while others believe they are practically without value. Neither of these extreme views is correct. That miotics can hold the disease in check for long periods of time cannot be doubted, but they cannot cure it. They must be properly used, that is, the pupil must be kept contracted. For this purpose Posey prefers salicylate of eserin, beginning with a solution of $\frac{1}{16}$ grain (0.00648 gm.) to the ounce (30 c.c.), and gradually increasing the strength until, if the drug has continued to act favorably, at the end of three years the solution has a strength of 3 grains (0.195 gm.) to the ounce (30 c.c.). The author prefers pilocarpin, as it is equally efficient and less irritating; the strength should usually be twice that of the eserin solution. The nitrate of pilocarpin is preferred by some surgeons to the hydrochlorid. Instillation of a solution of adrenalin (1 : 1000), according to A. Knapp, is usually followed, in one to five minutes, by dilatation of the pupil. The tension may be unaffected; sometimes it is decreased; occasionally increased. Conjunctival irritation can generally be prevented if the solutions are always fresh and sterile, and if the conjunctival sac is frequently irrigated with a boric acid lotion.

Massage of the eyeball is of distinct advantage; it may be followed by improvement in vision and deepening of the anterior chamber. Apparently it assists the action of the miotics. Usually it is not possible to employ it in acute glaucoma, but in simple glaucoma and in eyes the subjects of mild attacks it certainly temporarily lowers the tension. It may be applied by means of various instruments (see page 290), that is, vibration massage or with the help of suction cup (suction massage). Just as good results can be obtained by simple massage with the fingertips. Unfortunately, the value of massage has been unduly magnified by irregular practitioners. It is useful only as an adjuvant to other well-recognized procedures, operative and medicinal, and is particularly important in the postoperative treatment of glaucoma. Strychnin and nitroglycerin should be given to patients with chronic glaucoma, especially the latter drug, if there is increased vascular pressure. *High-frequency currents* have been advised.

Iridectomy, in the author's opinion, in most circumstances continues to be the most satisfactory operative procedure in the treatment of *acute* glaucoma. It should be performed *early*, in the early stage if possible, while the excretory apparatus is still intact and *before the root of the iris is welded to the cornea.* General anesthesia should be induced before its performance, because the high tension of the eyeball somewhat nullifies the action of cocain. Much depends upon the exact position of the iridectomy, which is difficult of performance on account of the narrow anterior chamber, and no caution

should be omitted which will secure perfect quiet on the part of the patient.

In performing iridectomy for the relief of acute glaucoma the following directions should be borne in mind: If a keratome (Fig. 347) is employed, it should be entered through the sclerotic coat 2 to 3 mm. from the apparent border of the cornea, and, after the completion of the incision (see page 727), should be slowly withdrawn in order to prevent a sudden gush of aqueous humor and a too rapid reduction of tension, which might be followed by intra-ocular hemorrhage. If the anterior chamber is shallow the iridectomy is usually more easily performed, and with better results, if a Graefe cataract knife (see Fig. 394) is employed in the usual manner (see page 765). The excision of the piece of iris should be complete up to the periphery—*i. e.*, up to the ciliary border—and no portion of the excised iris must remain in the angles of the wound (compare with page 438). This is a much more important matter than the excision of a large piece of the iris—for example, one-fifth of it—as is usually advised. A comparatively narrow technically correct iridectomy yields satisfactory results. If the tension is very high, preliminary scleral puncture, as advised by Priestley Smith and Gifford, is a useful procedure. But it should be remembered that scleral puncture has been followed by intra-ocular hemorrhage (A. Knapp). T. Harrison Butler believes that suitable treatment should be instituted (miotics and other measures) until there is a reduction of the increased tension of the eyeball, as this method enhances the ease and safety of the operation.

A favorable result may be expected from iridectomy if the tension is lowered; an unfavorable one if this remains high. If there is a sudden rise of tension a short time after the operation, accompanied by severe pain, there is reason to suspect intra-ocular hemorrhage. Maddox recommends abstraction of blood from the nose, that is, *artificial epistaxis* prior to operation for glaucoma (also cataract) in dangerously plethoric individuals.

Section of the iris is sometimes followed by an extensive hemorrhage into the anterior chamber. A prolonged effort to get rid of this blood should not be made lest the trituration produce cataract. The blood will be absorbed, although it may take many days and even weeks before this is entirely accomplished.

The reforming of the anterior chamber is sometimes delayed as long as a week. Occasionally a day or two after the operation there is some slight rise of tension in the eye, which is of temporary character.

There is difference of opinion as to whether the eye should be bandaged or not after operations of this character. The author believes that not only should a bandage be applied for the first few days to the eye upon which the operation has been performed but also to the fellow eye; and that the one placed upon the affected organ should remain there until complete restoration of the anterior chamber has taken place. The eye which has not been operated upon should be kept thoroughly under the influence of eserin or pilocarpin during the

course of the treatment, because it is well known in acute glaucoma that iridectomy may be followed by a speedy outbreak of the same disease in the opposite eye. In most instances the iridectomy should be placed directly upward, so that the overhanging upper lid may cover the coloboma. Should the primary iridectomy fail to reduce the tension permanently it may be necessary to reinforce it with a cyclodialysis or an anterior sclerotomy (see page 733), or by performing a corneoscleral operation.

The author has expressed his faith in the efficacy of iridectomy in *acute* glaucoma. Col. Elliot and other surgeons decide in favor of corneoscleral trephining or other operations designed to establish permanent filtration in the *acute* as well as in the *chronic* forms of glaucoma.

If the eye which has not been subjected to operation has a decidedly shallow anterior chamber, and if there is a history of so-called prodromal glaucomatous phenomena, it should be submitted to operation as soon as the iridectomy wound in the opposite eye has firmly healed, certainly before the patient passes from skilled observation, because it is practically certain that it will be attacked like its fellow.

If the signs of impending glaucoma are not clear and the eye is nevertheless suspected, the mydriatic test suggested by Edward Jackson, Harlan, and Brailey, which consists of the instillation of a solution of homatropin and noting whether it produces any rise in intra-ocular tension or pulsation of the vessels of the fundus, may be employed. Should the test be positive, it would seem proper to perform at once what Treacher Collins has called a *preventive iridectomy*, or, following Col. Elliot's advice, a corneoscleral trephining. If this is declined or deemed inadvisable, the patient should use daily a solution of a miotic (strong enough to keep the pupil contracted), and be provided with a stronger solution to be used in an emergency—*i. e.*, during a sudden attack. If both eyes are affected, both should be operated upon, provided the conditions are suitable, at proper intervals; sometimes in acute cases operation on one eye must be followed immediately by operation on the other.

One of the complications which may follow the operation of iridectomy in glaucoma is the formation of a bulging scar at the seat of the incision, sometimes called a *cystoid cicatrix*. This is especially true if due care has not been taken to free the angles of the wound from adherent iris. On the other hand, in severe cases, this very cystoid cicatrix, by permitting a filtering of the liquids, has been regarded as a favorable condition. In this connection the modern operations for the relief of glaucoma, by means of which a *filtering* (*fistulous*) *area* is produced, must be considered. They are discussed on pages 734–741.

The treatment of chronic congestive (subacute) glaucoma is less likely to be followed by the brilliant results seen in acute cases; and instances are on record in which after the performance of an operation, entirely correct in its technic, the disease has not been stayed or malignant glaucoma (see page 439) has resulted. This is particularly true if

degenerative changes have occurred in the iris. Nevertheless, iridectomy or one of its substitutes, that is, corneoscleral trephining, or iridosclerectomy, offers the patient a much better chance than if medicinal measures are alone relied upon, and should be performed as early as possible.

There is much difference of opinion in regard to the value of iridectomy in simple glaucoma (chronic non-congestive glaucoma), and some surgeons doubt the propriety of its performance in this disease, and depend upon miotics and certain internal remedies—for example, strychnin and nitroglycerin. Statistical information indicates that in a limited number of the cases of simple glaucoma submitted to iridectomy the results are immediately unfavorable—that is, the disease is not only not checked, but rapidly progresses to blindness; in a small percentage of cases the disease remains stationary—that is, the iridectomy maintains the condition of vision which was present before the operation; in a certain number of cases there is temporary amelioration, but later slow advance of the disease—a rate of advance, however, that is slower than if operation had not been performed; in a comparatively small percentage of cases the operation is followed by perceptible and permanent improvement in vision. It must be remembered, however, that statistical information, no matter how carefully gathered, may be misleading; briefly, ordinary iridectomy in chronic glaucoma should yield to operative procedures designed to establish a permanent filtering or fistulous area.

In a certain number of cases (H. Friedenwald has collected 24, 18 of them being women) a perfectly smooth iridectomy is followed by *malignant glaucoma.* The symptoms which usually appear one or two days after the iridectomy are: marked increase in tension, obliteration of the anterior chamber, fixation of the coloboma, ciliary tenderness, chemosis of the conjunctiva, swelling of the lids, and rapid loss of vision. Hence Schweigger's advice to operate in chronic glaucoma affecting both eyes, first upon the one with the more advanced disease, even if it is blind, is followed by some surgeons. If no complication arises, there is reason to hope that iridectomy on the fellow eye will be followed by a normal healing process. Evidently, this rule cannot arbitrarily be followed. The treatment of malignant glaucoma consists in the instillation of eserin, or posterior sclerotomy, and the administration of large doses of salicylate of sodium (Friedenwald).

It is not entirely certain how iridectomy cures glaucoma. It has been suggested that this is accomplished by the removal of the portion of tissue which closes the angle at the anterior chamber; by the moderation of the blood-pressure in the iris (Exner); by the filtration of the fluids of the eye through the line of healing, which, for this reason, has been called the *filtration scar;* by the permanent drain which the cut surface of the iris affords, inasmuch as it is not closed by reparative processes (Henderson). The details of performing iridectomy and sclerotomy will be described in the chapter devoted to Operations.

The operation of *sclerotomy* has been used as a substitute for iri-

dectomy, but the weight of testimony in favor of the latter operation is sufficiently great not to make it a more desirable mode of procedure than iridectomy except in selected cases. Every iridectomy which is peripherally situated, and in which the knife enters through the sclera some distance from the apparent border of the cornea, is in itself a sclerotomy. It is useful as a supplement to iridectomy if the tension is not reduced, and may be employed in old blind glaucomatous eyes to relieve pain. According to the late Dr. de Wecker, sclerotomy, followed later by iridectomy, which can then be performed more correctly owing to the improved state of the eye, is preferable to a primary iridectomy.

In recent years a number of important and usually satisfactory operations have been devised for the purpose of maintaining a *filtering cicatrix* or *area*, and, in so far as our present knowledge enables an opinion to be formed, their permanent results, other things being equal, are much more satisfactory than the simple iridectomy of former times. Of these, the most important are *Lagrange's operation*, by means of which an iris-free filtering cicatrix is produced with a combined iridectomy and sclerectomy (see page 734), *Herbert's wedge-isolation operation* (see page 735), *trephining the corneoscleral border* (Elliot's operation, see page 737), and *sclerectomy with punch-forceps* (Holth, see page 736); *iridocleisis* and *iridotasis* (page 741). The value of Heine's operation, or *cyclodialysis* (see page 741), in the treatment of glaucoma has been much discussed in the past few years; but it is evident that while it has its uses it cannot replace iridectomy or the operations just referred to. For further discussion on the comparative value of these operations see pages 734–742.

The operation of *sympathectomy*, or excision of the superior cervical ganglion of the sympathetic, for the relief of glaucoma has been performed a number of times. Little is heard of the operation at the present time, and evidently it has, very properly, failed to secure a permanent place in ophthalmic surgery.

For the relief of the pain of absolute glaucoma *opticociliary neurotomy* has been performed, and is still advocated by some surgeons. In the opinion of the author, enucleation or one of its substitutes is a better operation, but he also has had some excellent results with cyclodialysis and from corneoscleral trephining. In these circumstances, if the other eye shows any prodromal signs of glaucoma, it would seem proper that an iridectomy or a corneoscleral trephining should be performed in anticipation of the glaucomatous attack.

The association of glaucoma with various constitutional defects and disturbances of metabolism has been discussed (page 425). Therefore in the treatment of the disease local medication and operative procedures are not sufficient; each glaucoma patient should be thoroughly examined from the *general standpoint* and remedies and dietetic regimen ordered according to the findings. Lagrange's warning that glaucoma represents a "sick eye in a sick body" must not go unheeded.

Secondary glaucoma, or that form which arises in consequence

of some pre-existing disease of the eye, may, like the primary variety, assume an acute or chronic type.

It may follow inflammation of the iris and ciliary body with the production of extensive annular synechiæ; serous cyclitis, ulcers of the cornea which have perforated this structure and produced adherent cicatrices or staphylomatous bulging; swelling of the crystalline lens after needling; discission of after-cataract, and, occasionally, primary extraction of cataract; dislocation of the lens; detachment of the retina, associated with severe hemorrhage; the growth of a choroidal sarcoma or other intra-ocular tumor; cysts and tumors in the angle of the anterior chamber and choroidoretinitis, plastic choroiditis, thrombosis of the central retinal vein, and disease of the retinal vessels. Mayou calls attention to the early formation of vessels in the iris in glaucoma as a sign of thrombosis of the central retinal vein and to the dangers of operation in these cases because it may be followed by subchoroidal hemorrhage. Owing to injury the vitreous may pass forward into the anterior chamber, block the channels of exit, or press against the iris, and thus cause secondary glaucoma. Adhesion of the lens capsule to the cornea following traumatism or cataract extraction not infrequently results in secondary glaucoma (see also page 776).

In most of the instances mentioned there is no difficulty in diagnosticating secondary glaucoma by the history of the case and the knowledge of the pre-existing disease. This is not so easy if the original trouble has been deep in the eye, for example, a sarcoma. In these cases the glaucoma is usually absolute.

Treatment.—Secondary glaucoma, in general terms, requires the same treatment as the primary form of the disease, which must be modified according to the surrounding ocular conditions. A dislocated lens, or a lens swollen after discission for cataract, should be removed. Absolute glaucoma associated with great pain, if there is any suspicion of intra-ocular growth, indicates excision of the globe.

Hemorrhagic glaucoma is one type of secondary glaucoma in which numerous retinal hemorrhages appear as the result of thrombosis of the retinal vessels, or hyaline degeneration of their walls, or other causes likely to produce extravasation of blood (renal retinitis). The tension rises and the character of the disease may be acute, subacute, or chronic. This condition should be sharply differentiated from primary glaucoma associated with retinal hemorrhages, although sometimes it is exceedingly difficult to decide whether the glaucoma is secondary to the hemorrhages, or whether the hemorrhages have been produced by alterations in the tension of a glaucomatous eye. With the ophthalmoscope one may recognize the ordinary appearances of glaucoma and numerous retinal hemorrhages; or, in addition, there may be the lesions of the disease which have caused the hemorrhages and the glaucoma which followed them. Hemorrhage into the vitreous may occur, obliterating the fundus reflex; the cornea is steamy, the anterior chamber obliterated, the iris discolored, and the eyeball intensely injected and very hard.

Iridectomy is not usually followed by good results in hemorrhagic glaucoma, as it may be followed by intra-ocular hemorrhage. If attempted it should be preceded by posterior sclerotomy. Corneoscleral trephining has been tried with success; cyclodialysis has been recommended and the author has had some favorable results after the procedure. The results of anterior sclerotomy are more favorable than those of simple iridectomy. Posterior sclerotomy (tapping the vitreous) alone may be followed by relief, and paracentesis of the anterior chamber has been advocated. If the pain becomes intense and blindness ensues, enucleation is required. General treatment is of importance, as the patients are usually the subjects of vascular disease and high arterial tension; the cautious use of cardiac sedatives, nitroglycerin, and strict regulation of the diet and mode of life. Locally, measures to relieve ocular congestion and the miotics may be employed; dionin and holocain often afford decided relief.

Complicated Glaucoma.—Two kinds of complicated glaucoma are described which may be looked upon as varieties of the secondary form of the disease, namely, *cataract with glaucoma* and *high myopia with glaucoma.* In the former condition one eye alone is usually affected. It should be distinguished from the lenticular opacity produced by absolute glaucoma. During the formation of cataract glaucoma may occur, due probably to swelling of the lens and lessening of the circumlental space, and it is important to test frequently the intra-ocular tension of patients with developing cataract; preliminary iridectomy may be required. Glaucoma which develops in association with absorptive changes in a senile cataract has been attributed to toxic products brought into existence by this process (H. Gifford).

In high myopia with glaucoma the usual changes in the field of vision and the papilla are present. In addition to this there is more or less choroidal disturbance, which may itself be the cause of the glaucomatous condition. According to Hotta, the first change produced by the increased tension is an excavation of the nerve-head, and subsequently an ectasia into the intervaginal space of the immediately surrounding sclera. The *relative frequency* of myopia in glaucoma has been referred to (page 425).

Hydrophthalmos (*Hydrophthalmos congenitus; Keratoglobus; Megalocornea; Buphthalmos; Glaucoma Congenitum*).—In this affection there is slow but progressive enlargement of the eye in all its diameters; the cornea is flattened, the pupil dilated and sluggish, the iris atrophic and sometimes tremulous, the sclera thinned and of a bluish color, and the anterior chamber deepened; the intra-ocular tension is raised. The refraction is myopic, but not to as great a degree as the elongated axis of the eyeball would suggest, because, as Parsons, who has studied the subject most thoroughly, points out, the flattening of the cornea, the flattening of the lens, and displacement backward of the lens counteract the axial myopia. In the course of time the cornea may become cloudy (*keratoglobus turbidus*), although this is not usually the case (*keratoglobus pellucidus*). The papilla may be and usually is deeply

cupped. *Fissures in Descemet's membrane* may arise and appear as lines of grayish color with double contour, visible to the corneal microscope. They are a characteristic feature of the disease and may be well studied by means of the slit lamp.

The affection appears at birth or shortly afterward, and its incipient stages are believed to be intra-uterine. The precise cause is not accurately determined. Buphthalmos is more frequent in negroes than in white children, perhaps, as Zentmayer suggests, on account of the greater frequency of congenital syphilis among them. It has been ascribed to an intra-uterine iridokeratitis with increased intra-ocular tension; in other words, a form of congenital glaucoma. Pyle divided the disease into two classes: true hydrophthalmos, depending upon congenital defective development of the cornea, iris, or filtration channels, and hydrophthalmos secondary to fetal intra-ocular inflammation.

The *prognosis* is unfavorable; the affection often progresses to blindness. Iridectomy has been practised sometimes with a good result, more often with poor success, and, on the whole, is not advisable; some favorable results from repeated sclerotomies have been reported; indeed, Haab states that if these procedures are begun early enough, infantile glaucoma can be cured. Subconjunctival paracentesis has been advised. Corneoscleral trephining has been recommended and practised with encouraging results (Calhoun, Zentmayer, Bruns, Elliot). The technic, as Elliot points out, of corneoscleral operation in this disease differs in some respects from that in ordinary glaucoma (page 737). The author has observed beneficial effects from this procedure. Eserin or pilocarpin should be used.

CHAPTER XII

DISEASES OF THE CRYSTALLINE LENS

Congenital Anomalies.—In addition to congenital cataract and congenital displacement of the lens, which are described on pages 455 and 467, two anomalies require mention:

1. **Coloboma of the Lens.**—This defect is usually associated with a similar defect in the iris and choroid. The normal, rounded margin of the lens is replaced by a straight margin in a horizontal direction or incurved. The amount of the defect varies from a slight indentation to about one-quarter of the lens substance. It is almost always situated in the inferior half of the lens. A defect in the zonule of Zinn has been recorded (*coloboma of zone of Zinn*); also a general smallness of the lens (*microphakia*).

2. **Lenticonus.**—*Posterior lenticonus* is an abnormal curvature of the posterior surface of the lens or an anomaly of the nucleus (L. Müller), either unilateral and associated with lenticular opacities, or without such association, and then usually bilateral. With the plane mirror a sharp red disk, surrounded by dark shadows, like an oil-globule in water, may be seen (H. Knapp). *Anterior lenticonus* is a rare anomaly, and may be bilateral, as in a case studied by the author and Meyer Wiener, where each lens presented a pronounced cone, the tip of which almost touched the posterior surface of the cornea (Fig. 207). Verhoeff suggests a cone of this character may be due to persistence of the conical shape of the embryonic lens vesicle or to delayed separation of the lens from the cornea. According to Tscherning anterior lenticonus and shallowing of the anterior chamber take place during the act of accommodation.

Congenital aphakia, in association with the faulty development of the anterior part of the globe, has been reported. Aphakia may also result from the absorption and degeneration of a previously formed lens.

Cataract.—Under the term *cataract* are included several types of an opaque condition of the crystalline lens, of its capsule, or of both these structures, which anatomically are distinguished by the titles *lenticular*, *capsular*, and *capsulolenticular*.

Varieties of Cataract.—(1) *Primary;* (2) *secondary* to disorders in other portions of the eye; (3) *symptomatic* of a general malady or local injury.

A cataract is either *partial* and stationary, or progressive and becomes *complete*, and clinically is classified as *senile*, subdivided into *nuclear* and *cortical;* or, according to Axenfeld, *subcapsular*, *supranuclear*, and *nuclear; juvenile* or *presenile; congenital*, subdivided into *complete* or *partial; secondary* or *complicated; traumatic*, and *after-cataract.*

Cataracts are also classified according to their consistence as *hard*, *soft*, or *fluid*, and sometimes are designated by their color as *black*,

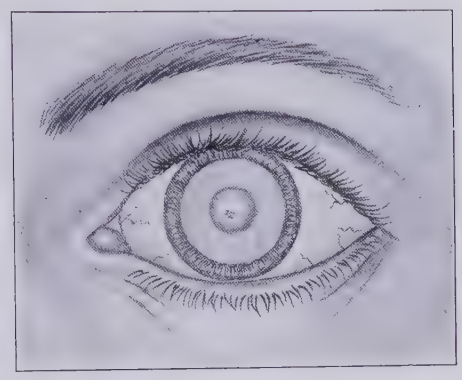


Fig. 206.—Posterior lenticonus (from a patient in the University Hospital).



Fig. 207.—Anterior lenticonus (from a patient in U. S. General Hospital No. 30).

white, *amber*, etc. *Blue* cataracts are occasionally encountered (*cataracta cærulea*). Although in many instances the precise division of cataract into special varieties may be unimportant, the following table, compiled

from the classifications employed in various standard works, may be useful to the student:

- Anatomically
  1. Lenticular.
  2. Capsular.
  3. Capsulolenticular.
- Clinically
  1. Senile — (*a*) cortical, (*b*) nuclear — general.
  2. Juvenile or presenile.
  3. Congenital
     - (*a*) complete: complete; congenital.
     - (*b*) partial: lamellar, or zonular; axial, or coralliform; punctate; discoid; pyramidal, or polar.
  4. Complicated or secondary: anterior polar cataract; posterior polar cataract; anterior cortical cataract; posterior cortical cataract; complete cataract.
  5. Traumatic.
  6. After-cataract.

Symptoms.—The following symptoms are present with more or less constancy in cataract, exemplified by the senile form of this disease:

1. *Change in Visual Acuteness.*—The amount of depreciation of sight depends upon the situation and extent of the opacity, and sometimes upon alterations in the refractive power of the lens. Thus there may be an increase in the index of refraction of the crystalline lens as an accompaniment of structural change in advancing years, causing myopia, often called *prodromal myopia.* In these circumstances distant vision is improved by concave lenses and reading becomes possible without the aid of convex glasses. This is the so-called "second sight." Such changes in the lens may cause a halo to appear around a light, for example, an electric light bulb. According to Landolt this halo is not visible around all lights indiscriminately, but around such as are situated at a given distance. It is due to the dispersion which luminous rays undergo in the equatorial region of the crystalline lens; the halo disappears if the light is looked at through a pin-hole disk (see also page 422). Changes in the lens may produce an irregular astigmatism, or an astigmatism "against the rule" may develop. As Edward Jackson insists, visual impairment due to cataract cannot be ascertained until the refraction of the eye has been corrected as accurately as possible.

2. *Hyperemia of the Conjunctiva.*—This is caused by the strain which the effort to see through a somewhat clouded lens produces.

3. *Pain and Photophobia.*—These symptoms are not prominent; but sometimes, owing to the condition of disturbed choroid which commonly is associated with cataract, patients complain of dull, aching pain or other asthenopic symptoms. Tinted glasses relieve the photophobia and permit slight dilatation of the pupil, which sometimes improves vision if the opacity is central. Pain, with rise of tension on

account of swelling of the lens, occasionally occurs. Indeed, *acute glaucoma* may be caused by this swelling of the lens during the formation of cataract, and the state of the intra-ocular tension deserves close attention in all patients with formed or forming cataract.

4. *Polyopia* and *monocular diplopia* are occasionally the result of incipient cataract, and are due to the irregular astigmatism which the alterations in the lens have produced.

5. *The Anterior Chamber.*—This may be normal in depth—the usual condition in incipient and mature cataract; shallower than normal—indicating a swollen lens; or abnormally deep—a symptom of a small lens.

6. *The Pupil.*—This may be natural in appearance and the mobility of the iris entirely normal; but sometimes exclusion of light or the instillation of a mydriatic fails to dilate the pupil.

We speak of the "color of the pupil." and this varies in cataract according to the degree of maturity and the hue of the opacity. Hence in the unilluminated pupil no change is seen in its color in incipient cataract; but in a ripe cataract the pupillary space may appear dull, gray, and even white, according to circumstances. In examples of so-called "black cataract" the pupil is dark. The mere inspection of the pupil, however, without optical aid is not sufficient to ascertain the condition of the lens, which continues to increase in size even with advancing years, if it remains clear. But it becomes firmer, straw colored, and reflects more light. This creates a dull sheen in the pupil which may be mistaken for cataract. The yellowish tint of the lens in advanced life may modify the relative perception of colors, which, indeed, may be so pronounced as to create *blue-blindness* (C. von Hess).

Diagnosis.—Since the introduction of the ophthalmoscope, the *catoptric test* has fallen into disuse, although it may be employed to determine the presence of the lens and in the diagnosis of black cataract.

This test is performed as follows: If, in a dark room, a lighted candle is moved before a healthy eye with dilated pupil, three images of the flame will be seen: two erect, formed by reflection from the convex cornea and anterior surface of the lens, the former producing the brighter image and the latter the more diffuse; and one inverted, relatively clearer, from the posterior surface of the lens. If the lens is opaque the inverted image is wanting, the deeper erect image also disappearing when the opacity involves the capsule, the corneal image being then alone visible.

Before using the ophthalmoscope for the detection of cataract the pupil should be dilated, preferably by instilling a solution of homatropin, cocain, or euphthalmin. The examiner proceeds in the manner described on page 107, and should detect in incipient cataract spots or streaks of opacity, often radiating from the periphery toward the center, which appear black from the interference with the reflection of light from the choroid. In like manner the nucleus may be seen to be hazy and the periphery clear, or the sectors of the lens are strongly marked. The beginning of cataract is also made evident by flaws in the lens, which

have been compared to cracks in glass, and are known as "striæ of refraction." If the entire lens is opaque, no portion of the pupillary space exhibits any red reflex from the fundus, although a lens which appears completely cataractous through the undilated pupil may exhibit spots of incomplete opacification in the periphery recognized by the transmitted red glare when the pupil is dilated. The final examination with transmitted light should be made with a +16 D lens or with a corneal loupe.

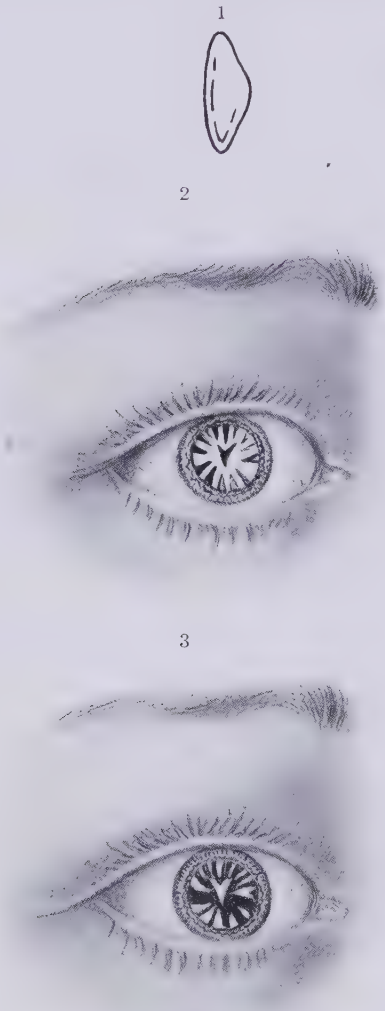


Fig. 208.—Cortical cataract: 1, Section of lens, opacities beneath the capsule; 2, opacities seen by transmitted light (ophthalmoscope mirror); 3, opacities seen by reflected light (oblique illumination). (Modified from Nettleship.)

With *oblique illumination* (see page 52) the opacities, if incipient, appear as white or gray streaks and dots.

When a progressive senile cataract is fully matured its presence may often be detected without any special examination except in the instances already mentioned, but it is a matter of the utmost importance to ascertain when this full maturity has been reached, or, in other words, whether the cataract is *ripe*. This is determined in the following manner:

The patient being placed in the proper position, the pupillary space is illuminated. If the opacity is complete, the opaque lens, covered by its capsule, is level with the margin of the pupil, and there is no shadow; if not, the major portion of the opacity is at a level posterior to the plane of the pupil, or, in other words, a clear or partly clear space is present between the iris and the opaque portion, and a dark semicircle appears upon the opacity at the side from which the light comes. This is the shadow of the iris. Shining sectors or the transmission of a red glare indicate immaturity, even if the shadow

is absent. In hypermature cataract the shadow is visible, but the surface of the lens is flat.

Development, Course, and Pathologic Anatomy of Cataract. — In progressive *senile* or, as it is sometimes called, *simple* cataract there is a period of growth from *incipiency* to full *maturity* which varies considerably, and ordinarily consumes from one to three years. Often the rate of increase is very slow, and immature cataract, especially of the cortical variety, may remain unchanged for many years. At other times the development of the disease is comparatively rapid. This slow progress of cortical senile cataract should be remembered, and the discovery of striæ in the lens need not condemn the patient to rapid deterioration of vision. Indeed, certain lenticular opacities remain practically stationary for years.



FIG. 209.—Nuclear cataract: 1, Section of lens, central position of opacity; 2, appearance by transmitted light; 3, appearance by oblique illumination. (Modified from Nettleship.)

The opacities begin either *equatorially—i. e.*, at the edge of the lens —or *centrally—i. e.*, at the nucleus. In the former case the striæ begin just beneath the capsule and are seen both in the anterior and posterior cortex. Sometimes broad sector-shaped opacities form, or the opacities may be narrow, or they may appear as delicate radiating lines. They gradually radiate toward the center (encroach on the pupil space), the nucleus becomes hazy and sclerosed, the cortical layers become swollen, more opaque, and, finally, the cataract is complete. Cataract, other things being equal, is more prone to begin in the lower part of the lens than elsewhere (see also page 450).

In the second variety the nucleus becomes hazy and the surrounding cloudiness always remains the most opaque portion of the cataract, which gradually spreads to the cortex (Fig. 209). Where diffuse clouding occurs first at the central portion of the lens, that is, in the layers immediately surrounding the nucleus, marked depreciation of vision is an early symptom.

A frequent lesion is a disk of opacity with irregular borders in the posterior layers of the cortex, web-like in structure, or sometimes looking like a collection of minute bubbles and striæ. This also causes marked disturbance of vision.

To a ring of opacity near the equator of the lens and behind the iris, seen in old persons and frequently stationary for long periods of time, the name *arcus senilis lentis* has been given.

Cataract may also begin as a more or less diffuse clouding or in the form of small dots, or blister-like bodies, scattered through the cortex, or located either in the anterior or posterior cortex, or in opacities which, with transmitted light, resemble dark flocculent precipitates. In the last-named circumstances the advance is more rapid than where the striæ are the first manifestation. Instead of going on to maturity, a nuclear haze or a spear of opacity may remain stationary, or at least show no practical change for years. Very exceptionally an anterior capsular opacity, not to be confused with such an opacity as occurs in hypermature cataracts or as is described on page 461, precedes the development of lenticular cataract by a long period of years.

Slit-lamp microscopy furnishes a method of investigating the manifold lenticular changes incident to the various stages of cataract formation. Indeed, investigations with the aid of this method have materially modified our knowledge of the development of cataract and the manifestations of the early and late lesions. We know that with slit-lamp and sunlight illumination (page 100) and the corneal microscope some kind of opacities may be detected in practically every crystalline lens. In many cases these opacities do not interfere with vision. Obviously, it is not possible to do more than refer to a few of the important observations. We are especially indebted to Vogt, Koeppe, and Gjessing for studies in this respect, and in this country to H. Gradle, R. von der Heydt, E. Blaauw, S. Gifford, and A. J. Bedell.

Coronal cataract (*"koronarkatarakt," Vogt's cataract*) is comparatively frequent after the age of puberty; prior to that period it is rarely found. According to Vogt it begins in the lens periphery in a wreath-like zone in the form of rounded or oblong opacities which are situated in the area of "the nuclear equator," in front of and behind it. A blue or bluish-green color is evident. In the course of years these opacities increase in number. If the process is progressive it may be regarded as one which ultimately produces senile cataract. Groups of shagreen-like globules are visible in elderly persons and in cataractous lenses situated in the intermediate lenticular zone (Gjessing). *Fissures* filled with fluid are visible in the anterior cortex (*aqueous clefts*), usually first in evidence about the fiftieth year of life. They are also found in the nucleus (Gjessing).

They are an indication, although not a constant one, of a tendency to cataract formation. Examining 111 "senile" eyes (nearly all patients over fifty years of age) C. E. Pfeiffer found 29.9 per cent. of aqueous fissures usually associated with lens opacity.

The various stages of cataract development are often classified by systematic writers into: (*a*) the stage of incipiency (early cataract); (*b*) the stage of swelling (intumescent cataract); (*c*) the stage of maturity (ripe cataract); (*d*) the stage of hypermaturity (overripe cataract).

During the formation of cataract the following changes occur in the lens: First, there is a separation of the lens-fibers with a collection of fluid between them, which coagulates into drops—the spheres of Morgagni. Later there are swelling, clouding, and fatty degeneration of the cortical fibers and the formation in them of nucleated vesicular bodies. Ultimately there is disorganization of the fibers, and the lenticular tissue is changed into fat-drops, spheres of Morgagni, and albuminous liquid, and the cortex separates from the capsule, the liquor Morgagni collecting between them. The lens nucleus becomes sclerosed, but in other respects is not greatly altered. Paul Römer has studied the pathogenesis of cataract from the point of view of serum investigations, and believes it is possible that, as a result of the degenerative processes of old age, antibodies are liberated in the blood which possess a definite affinity for certain parts of the lens protoplasm. They, by uniting with corresponding receptors of the lens protoplasm, are able to damage the lenticular cells, just as blood-corpuscles are destroyed by the union with them of specific cytotoxins.

Maturity may be succeeded by the stage of "overripeness" and the cataract gradually shrinks to a flat disk, or the later liquefaction of the cortical matter permits displacement of the nucleus, which may be brown, yellow, or quite dark in color. Examination fails to reveal striæ or sectors; whitish dots may be visible. Still later the cataract has a uniform white appearance, sometimes a slight bluish tint, a type which is known as *morgagnian cataract.* Tremulousness of the iris is seen in overripe cataracts. *Spontaneous rupture* of a morgagnian cataract has been reported (Gonzalez). Sometimes calcareous degeneration in the lens or its capsule may take place and a patch of capsular opacity is not uncommon in old and sometimes overripe cataracts.

The cataract, the development of which has just been described, is, for the most part, *hard—i. e.*, the nucleus of the lens is large. Under the age of thirty-five all cataracts are *soft—i. e.*, the nuclei are small or wanting, just as the lenses in which they develop have failed to attain the density which later they assume.

Causes of Cataract.—1. *Age of Life.*—Cataract which becomes complete is especially frequent after fifty years; but, as Fuchs remarks, it cannot be regarded as a physiologic attribute of old age. It is a pathologic process, and age, while it is an important factor in its development, must often be regarded only as a predisposing cause. Occasionally total cataract without apparent constitutional disease is found in adolescents. The beginning of cataract is not entirely peculiar to

old age, but may appear between the twentieth and the thirtieth year as an equatorial opacity. (See also page 450.)

2. *Sex.*—This appears to have no decided influence, the sexes being about equally affected, unless it be in the zonular variety, in which a greater liability of females has been recorded.

3. *Disease.*—Sugar has been found in the urine of about 1 per cent. of cataract cases. According to Klein, posterior polar, combined with posterior cortical cataract, unassociated with choroiditis, is significant of diabetes. Schanz's experiments indicate that the presence of sugar and acetone aid the action of light in causing cataract. A rapidly forming cataract in young persons who are the subjects of well advanced diabetes is the one best described as a *diabetic cataract.* Naturally, many elderly people who have glycosuria also acquire cataract, which, however, is not "diabetic" in the proper sense of that term. However, as Foster Moore points out, the frequency of cataracts "in older diabetics is greater than in other patients of the same age without diabetes." Albumin is present in about 6 per cent. of the cases, but the etiologic relation of nephritis to cataract has not been proved. Grilli's researches lead him to believe that cataract is caused by a species of dehydration of the lens, brought about by an insufficient elimination of solids by the kidneys, and a consequent rise of osmotic tension in the blood and aqueous humor. Doubtless, changes in the osmotic pressure of the fluids circulating around the lens, whether they are due to toxic causes or local disease, contribute to the formation of cataract, although Römer has shown that they may vary greatly without affecting the lens.

Cataract has also been noted in connection with idiopathic fevers and allied diseases, with gout, goiter, malaria, influenza, rachitis, syphilis (Bos), angiosclerosis, and especially atheroma of the carotid (Michel), epilepsy and other convulsive seizures, meningitis (Bock), certain cutaneous affections (Mooren, Rothmund, Andogsky—*dermogenetic cataracts*), with bronchocele, with hookworm disease (Calhoun), and sea-sickness (Weeks). A connecting link, however, in many instances, between constitutional maladies and opacities of the crystalline lens has not been established. The frequency with which lenticular opacities, either cortical or at the posterior pole, appear in eyes the retinal arteries of which show signs of degeneration (see page 519) is well known. It is possible that sclerotic changes in the nutrient vessel of the anterior uveal tract may aid in the development of lenticular degeneration. Schiötz has described a relationship between cataract and disturbances of internal secretion. The association between myotonia atrophica and cataract (opacities deep seated and irregular [L. Paton]) has been noted. They and the myotonia are probably due to the same metabolic defect. J. Fischer and O. Trienenstein investigating a number of senile and presenile cataract patients to discover evidences of tetany, or latent tetany, found such evidence in 88 per cent. of the cases; also premature grayness of the hair, dryness of the skin, etc. Peters has also attributed some nuclear cataracts to the toxin of tetany. H. Kirkpatrick believes that the cause of primary cataract is a constitutional

one; it may follow thyroid and parathyroid deficiencies. The influence of abiotrophy in relation to cataract has been emphasized by Treacher Collins. Frequent exposure to light rich in ultraviolet rays may be responsible for lenticular changes. It is possible that such rays after diffusion in the lens reach the ciliary body where they cause disturbances of nutrition which may be responsible for cataract (Van der Hoeve).

4. *Occupation.*—Cataract is especially frequent among glass-blowers, and is attributed to the effect of the radiated heat and excessive perspiration. In *bottle-makers'* or *glass-workers' cataract* the lesions often consist of a dense, well-defined disk of opacity in the center of the posterior cortex, surrounded by smaller opacities. These cataracts, according to an investigation of the Royal Society of England, are not caused by the luminous rays and not directly by the ultraviolet rays. Heat is probably the active agent, affecting the lens either directly or indirectly by disturbing its nutrition. It is not improbable that additional investigations such as the author made years ago would show the same liability to cataract in puddlers and others exposed to intense heat; indeed, Cridland has observed in furnace-workers cataract resembling that which is frequent in glass-blowers, to which the name *furnace-workers' cataract* has been given. For those forms of cataract due to prolonged radiant heat (glass-workers, puddlers, gold smelters, etc.) Cridland has proposed the descriptive term *ray cataract.*

5. *Heredity.*—Remarkable examples of the influence of heredity in the formation of cataract have been published. It has been noted that the tendency is more marked in the child-bearing period, and that the transmission is through the female line; transmission through the male line only, however, has been recorded. All phases of this subject were elaborately studied by the late Mr. Nettleship. In both groups of acquired cataract, that is, senile and presenile or juvenile cataract, the transmission is almost always direct. Occasionally a generation is skipped. Referring to the frequency of *inherited cataract*, he found that women are somewhat more liable to familial acquired cataract than men. In *cataract families* the lenticular opacities may appear among the descendants at the same age, or the succeeding generation may be affected earlier than the preceding one (anticipation). E. Treacher Collins assumes there must be some inherent defect in the vitality of the lens fibers, which results in their degeneration, to explain hereditary postnatal cataract, where often many members of a family are affected at comparatively an early age. For such cataracts he suggests the name *abiotic cataract.*

6. *Toxic Agents.*—Cataract has been produced artificially by poisoning rabbits with naphthalin (*naphthalin cataract*), sodium nitrate, and other toxic agents. In addition to the cataract, there are changes in the retina and vitreous and also general disturbances. It also occurs in workers in naphthalin.

During epidemics of *ergotism* patients are at times affected with cataract (*raphanic cataract*), the appearance having been noted almost

exclusively in the convulsive type of this toxemia; hence it is not certain whether the lenticular opacity results from the poisoning by the ergot or on account of the convulsions.

7. *Traumatism.*—Cataract may follow a *direct* injury to the lens, or be caused in an *indirect* manner—for example, by a concussion (*concussion cataract*) (page 460). To this category belong those cataracts which have followed a *lightning stroke.* A number of examples are recorded, both double and single, partial and complete. In addition to the cataract, optic neuritis, optic atrophy, rupture of the choroid, iritis, iridocyclitis, miosis, mydriasis, and palsy of accommodation have been observed. Cataract may also be caused by a violent *electric shock* and apparently has resulted from exposure to *x*-rays, while being used in the treatment of lupus of the lid (O. Wilkinson). *Electric cataract,* such as may be caused by a short-circuited current of high voltage, reveals to slit-lamp microscopy cork-screw-shaped opacities, and lace-like threads crossing each other and not following the lines of the lens fibers.

8. *Diseases of the Eye.*—Cataract may be secondary to numerous acute and chronic affections of the eye—viz., iritis, iridocyclitis, iridochoroiditis, choroiditis, detachment of the retina, glaucoma, and diseases of the cornea, especially sloughing ulcers. The frequent coexistence of disturbance of the choroidal coat and incipient cataract has led to the opinion that while opacity of the lens (so-called senile) is a condition commonly seen in advanced life, it does not, in all probability, depend upon senile changes, but upon local pathologic states involving the nutrition of the eye itself.

9. *Accommodative Strain.*—Investigations show that a large majority of cataractous eyes are hyperopic and astigmatic, and it has been stated that the danger of cataract is increased where the astigmatism is against the rule and remains uncorrected. The evident prophylactic measure is the use of proper glasses.

The etiology of cataract is by no means always clear, and often several factors are necessary to explain it; sometimes no direct cause can be assigned; frequently there are extra-ocular causes and the cataract results from nutritive disturbances.

The following additional facts in regard to the clinical varieties deserve attention:

I. **Senile Cataract** (*Simple Cataract; Gray Cataract*).—This, representing the type of general cataract which develops after the fortieth year of life, is nuclear, cortical, or mixed in its origin. It may not appear before the sixtieth year, but after that period of life lenticular changes which, however, do not necessarily advance rapidly, are exceedingly common. Its course from incipiency to full maturity has been described.

The color usually is gray, and the nucleus, which itself does not become cataractous in the usual sense of that term, but is sclerosed, may be recognized by its yellowish or brownish hue and its waxy appearance.

If the nucleus is small and the surrounding cortex uniformly white,

the cataract is comparatively *soft;* if the nucleus is large and the color of the cataract distinctly gray, yellowish, or brownish, it is *hard.*

Instead of a gray or grayish-white color the cataract may be yellow or amber, or the sclerosis of the nucleus extends to the cortical substance, so that the whole lens is brownish and the pupil black (*black cataract*). Occasionally *cholesterin crystals* may be found in cataracts, not only in the senile but also in the juvenile variety.

Senile cataract generally is bilateral, one eye being more affected than its fellow; but a cataract may attain maturity in one eye before the other lens is involved (*unilateral cataract*).

II. **Juvenile cataract** is a term descriptive of those opacities of the lens which occur before the fortieth year of life. To such cataracts the name *presenile* is also given (Nettleship).

In forms of cataract developed in early life the evidence of the influence of heredity is often strong.

General cataracts in young persons (*complete cataract of young people*) may arise without known cause or from one or other of the causes already recorded. These are bluish-white, often have a sheen like pearl, and are soft.

III. **Congenital Cataract.**—This may appear as a complete or partial opacity of the lens. In the *complete* form the lens usually is white or bluish-white in color, densely opaque, and *soft.* The eye may be otherwise healthy, or there may be changes in the choroid, retina, optic nerve (congenital amblyopia), and sometimes vices of conformation, as coloboma, microphthalmos, and hydrophthalmos. Disturbances of nutrition during intra-uterine life, changes in the choroid, arrest of development, consanguinity of parents, and heredity have been invoked to explain its existence.

There are several varieties of *partial* congenital cataract:

(*a*) *Zonular, lamellar,* or *perinuclear cataract* appears, as its name implies, in the form of an opaque layer surrounding the clear, but sometimes cloudy, center of the lens, and is the most frequent form of partial congenital cataract. On the surface streaks of opacity are often evident, which may project into the clear cortex and are called "riders." Usually it is double, but may be unilateral, and is either congenital or forms in early infancy. The cataract is stationary in most instances, but occasionally becomes complete.

If the center of the pupil is examined, a reddish point surrounded by a grayish halo will be observed. When the pupil is dilated with atropin and examined by means of the ophthalmoscopic mirror the central dark zone will be apparent, surrounded by a reddish circle, due to the reflection from the fundus passing through the peripheral part of the lens, which remains clear. With oblique light the appearances may be as in Fig. 210. A rare type is several zones of opacity separated by zones of transparency. Patients with zonular cataract act like myopes, and the refraction of the eye may be myopic. Macular changes are not infrequent.

The cause of lamellar cataract is not certainly known. In the

congenital variety it is probably due to some development defect; in the variety arising in early infancy some fault in nutrition has occurred. Most often the subjects are rachitic, and present the teeth and cranial asymmetry peculiar to this affection. Peters considers tetany a more common cause than rachitis, and Hesse and Phelps emphasize the frequent association of zonular cataract and tetany—both are attributed to the faulty calcium metabolism depending upon parathyroid insufficiency. A history of convulsions is common, and dental defects, which are present in the form of lines, furrows, or terraces, may lie transversely across the incisors or canines. Anatomically, lamellar cataract consists of a narrow zone of degenerative change in the lens-fibers, situated between the nuclear and cortical areas (Lawford).

Cataract may develop in later life, that is, between the ages of twenty and fifty, in persons who suffer from tetanic spasms. These patients may also suffer from loss of hair, necrosis of the nails, and chronic skin eruption. To such cataracts the name *tetany cataract* has been given, and they are apparently due to a toxin which affects epithelial structure (E. T. Collins) (see also page 452).

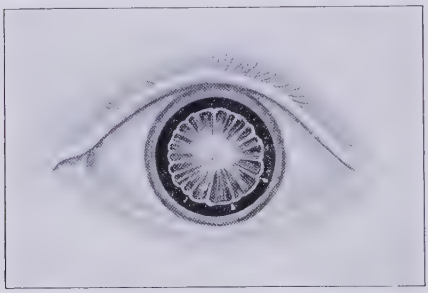


Fig. 210.—Zonular cataract (after Spicer).

(*b*) *Central cataract* (*central lental cataract*) consists of a white opacity in the central part of the lens, due probably to faulty development at an early stage of intra-uterine existence. Sometimes vision is surprisingly good; at other times it may be poor, and defects of development in the eye may be present and nystagmus may develop.

(*c*) *Pyramidal Cataract.*—This is also known as *anterior capsular* or *polar cataract*, and consists of a small, well-defined, pyramidal-shaped or circular opacity due to hyperplasia of the capsular epithelium and degeneration of the lens-fibers in that position. It probably arises in consequence of contact of the lens and cornea in fetal life, which causes an arrest of osmosis of nutritional fluid (E. T. Collins). Mules suggested that these cataracts may be cretified remains of the pupillary membrane.

At the posterior pole of the lens an opacity similar to the one described may be found, known as a *posterior polar* or *pyramidal congenital cataract*. It is caused by vestigial remains of the hyaloid artery at its lenticular attachment, or persistence of a part of the posterior vascular sheath of the lens, and, strictly speaking, is not a true cataract, that is, the changes are not in the lens. These opacities are sometimes separated into those which lie beneath the capsule and those which exist upon its surface. A small dot-like opacity of this origin, and which does not disturb vision, is quite common. S. L. Ziegler and

J. M. Griscom have published a report of *hereditary* posterior polar cataract (their two patients having had "double posterior polar cataract of the hereditary type"; there is no description of the appearance of the cataracts). A study of the pedigrees which they present shows there were 64 members in four generations, of whom 24 had congenital cataracts. The relative percentage of females affected was slightly higher than that of males. In no case did normal parents produce affected children.

(*d*) *Punctate cataract* is a form of congenital lenticular change in which the opacities present themselves in the form of more or less fine points, occupying the center of the pupillary space. These points or dots, however, may extend through the anterior cortex to the periphery of the lens, and, as in Holloway's case, have a bluish tint and be present in several members of the same family. *Punctate cerulean cataract* is usually non-progressive; small stellate figures are also visible. Forms of punctate cataract, which appear in childhood and in early adolescence, occur frequently in Mongolian idiots, well studied by Pearce, Rankin, and Ormond. Other types occur in older subjects—anterior and posterior opacities. W. M. Van der Scheer found 22 among 36 patients. The cataract remains stationary for a long time. It is not very uncommon to find in healthy adults punctate lesions in the lens, that is, in the anterior cortex, and in two patients under the author's care the lesions are disposed in the periphery as almost equidistant spots; such punctate spots are probably congenital in origin; they do not increase and do not interfere with visual acuteness.

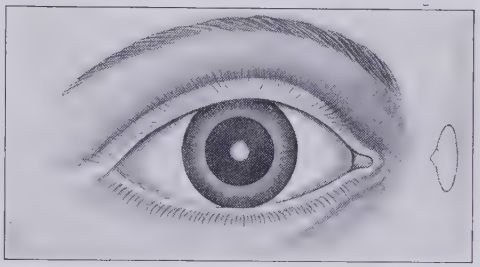


FIG. 211.—Anterior polar cataract (after Nettleship).

(*e*) *Fusiform cataract* is a rare variety characterized by an opaque stripe passing from the anterior to the posterior pole of the lens, sometimes with offshoots, disposed like coral branches. It may be combined with zonular cataract. It is also known as *axial* or *coralliform* cataract, and is prone to occur in families. Nettleship's list contains the record of one family in which 30 members, in four generations, were known to be affected. Microscopically, a lens with coralliform cataract shows numerous crystals most marked at the anterior and posterior poles; the surrounding lens substance exhibits vacuolization and fragmentation. The nature of the crystals is not definitely known; they probably represent crystallized lens proteins (Verhoeff). Nettleship and Ogilvie have described a peculiar form of congenital family cataract in which a disk of opacity (*discoid* or "Coppock" cataract), "consisting of a single layer, always thin, but varying in transparency, is situated behind the nucleus, but well in front of the posterior capsule."

A similar if not identical form of family cataract has been reported in this country by Burton Chance. Discoid and lamellar cataracts are essentially similar, the former being smaller and more deeply situated due to backward displacement of the nucleus. Disk-shaped

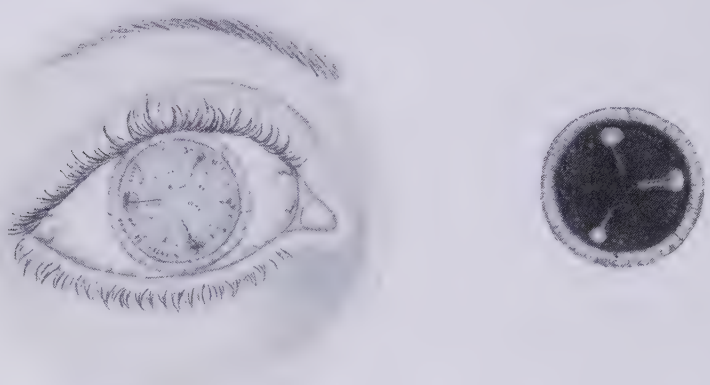


FIG. 212.—Congenital cataract of peculiar type (from a patient in the University Hospital).

opacities behind the nucleus, often hereditary, are also termed "Doyne's cataract."

IV. **Complicated or Secondary Cataract.**—This may be *complete* and arise in consequence of the various diseases of the eye enumerated on page 454. In iritis, for example, fibrinous exudations are attached



FIG. 213.—Coralliform cataract (courtesy of Dr. William Zentmayer).

to the lens-capsule, contraction occurs, the capsule is disturbed in its relation to the underlying lens-fibers, which are separated, and cataract forms. If this process is a limited one, the lenticular opacity may remain circumscribed. It may also be *incomplete*, and appears in the following varieties:

(*a*) *Anterior Polar Cataract.*—In addition to the congenital variety of this opacity there is an acquired type, which arises in consequence of a perforating ulcer of the cornea—for example, in ophthalmia neonatorum (see page 217). In infants' eyes it may follow ulceration of the cornea without perforation.

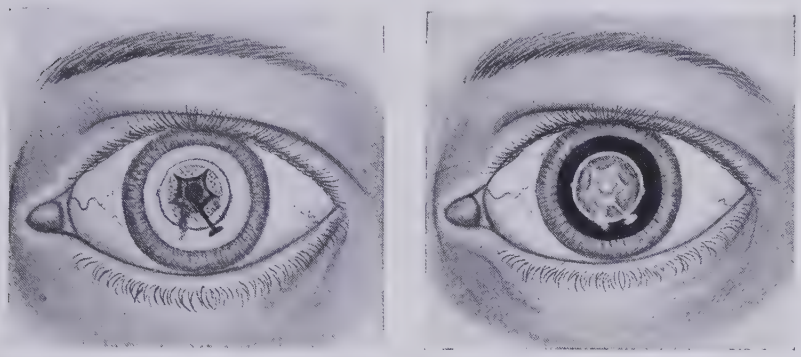


FIG. 214.—Unusual form of star-shaped opacities of the crystalline lens, probably congenital (from a patient in the University Hospital).

(*b*) *Posterior polar cataract,* as a congenital variety, has been described. As before noted, being outside of the lens system, it is not a cataract in the true sense of that term. In some cases, however, a posterior polar cataract, that is, one on the posterior surface of the posterior capsule of the lens, is combined with an opacity in the lens close beneath the capsule.

(*c*) *Anterior and Posterior Cortical Cataract.*—This form of cataract, usually in the posterior cortex and generally star-shaped or in the form of a rosette, is a common variety of complicated cataract, seen in high myopia, vitreous disease, disseminated choroiditis, and pigmentary degeneration of the retina. It may remain stationary for a long time, disturbing vision in proportion to its density, or it may progress and become complete. A similar appearance in the anterior cortex of the lens is sometimes visible; occasionally the opacity exists in both positions at the same time. When either anterior or posterior cortical cataract in these circumstances becomes total its extraction is indicated, but the prognosis is not as favorable as it otherwise would be because they are complicated with the ocular diseases which have been mentioned.

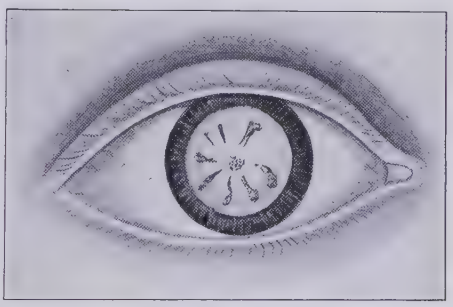


FIG. 215.—Posterior cortical cataract seen by transmitted light (from a case of pigmentary degeneration of the retina).

A form of complicated family cataract has been described by Purtscher and by Zentmayer. It occurs about the age of thirty in eyes with thin, bluish-white scleras, tremulous grayish-brown irides, deep anterior chambers, contracted pupils, and a tendency to glaucoma after operative interference.

V. **Traumatic Cataract.**—This is caused by *direct* injury to the lens by some penetrating substance which lacerates the capsule and permits the entrance of the aqueous humor which permeates the lens-fibers. (See also page 454.) The lens substance swells up, becomes opaque, and some of it may escape into the anterior chamber through the capsular wound. The clouding of the lens begins very soon after the injury. Absorption takes place in about six weeks, more rapidly in young subjects than in adults Occasionally the absorption is complete, but often some opaque layers or fragments of lens tissue remain within the healed capsule, that is, a traumatic after-cataract. This course represents the most favorable outcome of such an accident. In other cases there may be iritis, cyclitis, and secondary glaucoma, owing to swelling of the lens and elaboration of lens toxins. (See also page 353.)

Instead of going on to complete opacity, an injured lens, in some instances, presents a limited opacity, which remains stationary; in other instances this disappears, and in still others there is slow advance of the opacity. The complete disappearance of circumscribed opacities, formed after small wounds of the capsule, which speedily close, has been observed.

A more *indirect* mechanism of traumatic cataract is *concussion* (*concussion cataract*)—a blow upon the eye causing a slight rupture of the anterior or posterior capsule, followed by opacity, which may become general or retain a limited size for a long time. According to Nettleship, absorption of a complete concussion cataract is more uncommon than where the lenticular opacity has followed a direct trauma, although the lens may gradually shrink in size.

A *ring-shaped opacity* may appear on the surface of the lens after contusion of the eye, generally in young eyes. This is a circular opacity, 3 to 4 mm. in diameter, concentric to the margin of the pupil, and of a brownish color. The lesion is sometimes called *Vossius' ring*, or *contusion-lesion of the lens* (Caspar). Vossius ascribed the lesion to pigment adherent to the capsule or to disturbance of the epithelium. It is, as it were, a cast of the iris margin and its pigment on the capsule. This explanation is not, however, accepted by all observers. In several eyes studied by the author the lesion disappeared within a few weeks after the injury. Remote changes in the lens (fine punctate opacities and vacuoles) may take place and can be detected by means of the slit lamp (Vogt).

VI. **After-cataract.**—This name has been applied to those changes which occur in the capsule of the lens remaining after the extraction of cataract. It is usually called *secondary cataract.*

These changes may depend on proliferation and thickening of the capsular epithelium; on agglutination of the two layers of the capsule,

the anterior part being so folded over that it has retained cortical material, which has thus been shut off from the dissolving action of the aqueous and remains as a membranous opacity; or upon new formed tissue between the capsule layers of thickened elements from the anterior part of the vitreous. If there has been postoperative reaction, fibrinous exudation from the iris adds to the opacity. (Consult Fig. 401.) The name, that is, secondary cataract, has also been given to the dense white membrane (*membranous cataract*) which is composed of deposits of lymph, and fibrinous and plastic exudation, and to which the iris and even the ciliary processes are adherent, and which follows postoperative iridocyclitis.

VII. **Capsular Cataract.**—The name *capsular cataract* is applied to thickenings and proliferations of the capsular epithelium, and sometimes to subcapsular degeneration of the lens-fibers, which may be congenital, may follow inflammatory processes of the eye (corneal ulcer), and may occur in connection with other degenerations in overripe cataract.

VIII. **Capsulolenticular cataract** is the name applied to opacity of the lens associated with thickening of the surrounding capsule, most commonly in the center of its anterior portion.

Prognosis.—Incipient cataract in the form of striæ in the anterior cortex need not doom the patient to rapid deterioration of sight, because the existing vision is often maintained for long periods of time. *Spontaneous disappearance of senile cataract* has been reported. According to Pyle, this may occur on account of ruptured capsule, dislocation, or degenerative changes. Partial disappearance of immature cataracts in diabetics as the result of general treatment has been reported.

Operation is preferably deferred until the cataract is *ripe*. The surgeon must ascertain whether the eye itself is in a healthy condition to enable him to define the probable result of operative interference by attention to the following considerations:

(*a*) *The probable condition of the interior of the eye*, if no data of ophthalmoscopic examinations during the incipiency of the cataract are at hand. This is ascertained as follows:

Place the patient before a lighted candle or a small electrically illuminated bulb about 4 meters distant—the light should be distinctly recognized. This gives evidence that the macular region is free from extensive disease, but does not exclude a small lesion. Next cause the eye under examination to fixate the light attentively, and move a second lighted candle or electric bulb radially through the field of vision. The light should be recognized as soon as the rays strike the edge of the cornea, and the patient should be able to indicate the direction in which it is coming. Thus the "light-field," or the "projection of light," is tested.

If the patient fails to appreciate the candle-flame in any portion of the field, coarse changes may be suspected—*e. g.*, extensive choroiditis, detachment of the retina, glaucoma, etc.; but it is not possible to detect

a small area of central choroiditis by this means (see also page 394). The macular region should be investigated by requiring the patient to note the separation of two small centrally placed flames, or by causing him to look at the light through a small aperture in the center of a disk. Fluid vitreous, indicated by tremulousness of the iris, is an unfavorable sign. Should there be no light perception, the cataract is an unsuitable one for operation except that in certain circumstances its extraction is justified in order to improve the patient's appearance.

(*b*) *The Probable Condition of the Refraction.*—It may be impossible to ascertain this unless some record is at hand of an examination when the media were still clear. Some idea of the refraction is obtainable by examining the glasses which the patient may have used during his reading days. High myopia renders the prognosis less favorable; indeed, the vision after operation in myopic cases, other things being equal, is not so good as that of hyperopes.

(*c*) *The Mobility of the Iris; Its Reaction to a Mydriatic.*—This should be prompt and normal. Failure of iris reaction in either case may indicate imperfect conductive power in the optic nerve, or atrophy, or other change in the iris.

(*d*) *The Age and General Condition of the Patient.*—Advanced age alone does not militate, as much as it would seem likely to do, against successful cataract extraction. So, too, the extraction of diabetic cataract is usually followed by good results, although the tendency to postoperative iritis is probably greater in diabetics than in those who are free from glycosuria. The presence of chronic Bright's disease, while a complicating circumstance, does not forbid the operation. Great feebleness, dementia likely to become worse with confinement, nasopharyngitis, advanced arteriosclerosis, eczema, enlarged prostate and cystitis, and chronic bronchitis are unfavorable conditions. According to Hansell, syphilis should be regarded as a dangerous complication.

(*e*) *The Condition of the Area of Future Operation and of Its Surroundings.*—Disease of the lacrimonasal channels, trachoma, chronic conjunctivitis, and blepharitis contraindicate cataract extraction because the wound is almost certain to become infected by the unhealthy discharges. In such circumstances a line of treatment later described must be instituted before operation. A matter of importance, not always attended to, is the state of the rhinopharynx. This should be reasonably healthy to secure the highest type of success. The teeth and tonsils should be carefully examined for areas of focal infection. Eczema of the face or other regions of the body is a source of danger. Prior to cataract extraction a careful bacteriologic examination of the conjunctiva is important.

(*f*) *The Type and Condition of the Cataract.*—In making a prognosis the size of the nucleus and its position, the probable consistence of the cortex, the primary or secondary nature of the cataract, and its stage of maturity must be considered. Certain conditions (amblyopia) influence the prognosis in complete congenital cataract, and in the

partial varieties, like the lamellar form, the eye may be defective in construction. In traumatic cataract the extent of injury to parts other than the lens must be considered.

Treatment.—This may be divided into the treatment of *immature* and of *mature* cataract.

Drugs do not exist which can dissolve a growing cataract, and the use of electricity, which has been recommended, is of no value. Light massage of the eyeball after the instillation of a mixture of glycerin and boric acid solution has been recommended (Kalish); or a 1 per cent. solution of iodid of sodium or a ½ per cent. solution of iodid of potassium may be similarly employed. This procedure in some cases seems to afford a certain amount of relief, probably because of a stimulating effect on the anterior circulation of the eye.

1. The refraction should be carefully tested and that glass ordered which gives the most accurate vision. It may be necessary to make frequent changes in the correcting lenses to conform with the alterations in refraction brought about by alteration in the lens. Correction of *lenticular* myopia usually markedly improves the distant vision (see page 446).

2. Congestion of the choroidal coat may be relieved by the exhibition of certain alteratives, among which the iodids of sodium and potassium and syrup of hydriodic acid are the most suitable. The iodids may be combined with small doses of bromid of potassium or bromid of sodium. Biniodid of mercury has also been tried. Subconjunctival injections of iodid of potassium have been recommended (Badal, von Pflugh); of their value the author has no knowledge. Dor advocates the treatment of cataract locally with a solution composed of 4 grams of desiccated sodium iodid, 4 grams of crystallized calcium chlorid, dissolved in 500 grams of distilled water. The solution should be applied for half an hour a day by means of a glass eye-bath. Some observers report favorable results from the persistent use of dionin in solutions of gradually ascending strength, and favorable results have been reported; the remedy should be employed. Col. Henry Smith strongly recommends subconjunctival injections of cyanid of mercury (1 : 4000–6000). The treatment of incipient cataract by radium has been advocated by Martin Cohen and Isaac Levin.[1] The moderate use of the eyes may be permitted without danger of hastening the process of maturation. If the patient suffers from diabetes, nephritis, lithemia, or arteriosclerosis, suitable dietetic and medicinal measures should be employed. Drinking water freely is advised by Edward Jackson.

3. Often comfort may be given and vision improved by keeping the pupil dilated with a weak mydriatic (if the opacity is central). Tinted lenses, which correct any existing refractive error, should be worn. In other cases a miotic is useful.

If the vision of eyes suffering from cataract of the nuclear type is improved by mydriasis, this has been given as an indication for *optical iridectomy*, but is not a sufficient one unless the patient finds by observa-

[1] Transactions, Section of Ophthalmology, Amer. Med. Assoc., 1919.

tion that the increased visual acuteness, as noted by test-type examination, is also advantageous in pursuing his ordinary occupation.

Artificial Ripening.—The exceeding slowness with which a senile cataract may progress often leaves the patient in a state of semiblindness. To remedy this, several methods have been proposed for hastening the process of ripening.

Simple division of the anterior capsule; division combined with iridectomy (Mooren); division and external massage (Rohmer); iridectomy and triturating the lens-fibers by rubbing the cornea over the coloboma with a horn spoon (Förster's method); paracentesis of the cornea, and internal massage directly on the anterior capsule with a small spatula (Sasso and Ricaldi and B. Bettmann, of Chicago); and simple paracentesis of the cornea with external massage (T. R. Pooley, of New York, and J. A. White, of Richmond, an operation practised by the latter surgeon with much success).

A discission, after the manner of Graefe, carried deep into the lens substance, was recommended by Schweigger as the only satisfactory method, especially before the fortieth year; and a *preliminary capsulotomy* has been advised and practised by Homer E. Smith (see page 772). He has reported excellent results, as have some other surgeons who have followed his method.

Extraction of Immature Cataract.—Some operators of extensive experience hold that the usual criteria of ripeness are erroneous in that period when accommodation is annulled by physiologic changes in the lens—that is, about the sixtieth year—and the lens may be extracted safely even if it is in part unclouded. It may also be done successfully at an earlier age.

Many operators, following McKeown's advice, after the extraction of immature cataract resort to intracapsular irrigation and wash out tenacious cortical material with a suitably warmed physiologic salt solution. Indeed, irrigation of the anterior chamber forms part of the technic of all cataract operations in the hands of some surgeons. Suitable irrigation apparatus has been designed by McKeown and by J. A. Lippincott, but a flat-tipped glass tube to which is attached a rather large rubber bulb answers the purpose equally well.

If the unripe material is not removed it may swell up and cause iritis, probably because of development and liberation of toxins. Therefore the safest plan is to wait for maturity; but if this is impossible or very undesirable or the patient is unwilling to wait until the cataract is mature, the author has been in the habit of extracting an unripe cataract after preliminary iridectomy in preference to performing a ripening operation, and if necessary has employed irrigation with normal saline solution to get rid of cortical remnants which could not otherwise be satisfactorily expelled. He has practised deep discission or preliminary capsulotomy, but has practically abandoned these operations.

According to Colonel Henry Smith, Derrick Vail, W. A. Fisher, and a few other surgeons, the best means of managing immature cata-

ract is its *extraction in the capsule* by the so-called Indian or Smith method (see page 770). The stage of immaturity at which Smith advocates the extraction of immature cataract in the capsule is where the opacity has progressed so far as to unfit the patient for the performance of his ordinary duties. With this operation in this respect the author has had no experience. Immature cataracts can be removed equally well by other methods of intracapsular extraction (page 771).

Extraction of Mature and Complete Cataract.—*Mature* cataract requires an operation for its removal, differing according to the age of the patient and the consistency of the cataract.

Hard cataracts, or those which occur after the fortieth year, are suitably removed either by (*a*) simple extraction (extraction without iridectomy), (*b*) combined extraction (extraction with iridectomy), or extraction after preliminary iridectomy, (*c*) intracapsular extraction (extraction in the capsule), and (*d*) phacoeresis (removal by suction).

Soft cataracts, or those which occur before the thirtieth year, are suitably removed by (*a*) linear extraction, (*b*) the needle operation, or that of solution by discission, and (*c*) the suction method. A soft cataract before the twenty-fifth year may be removed through a linear incision into the cornea, and a semifluid one by suction.

Treatment of Congenital Cataracts.—According to E. Treacher Collins, a child should be ten months old before operating for congenital cataract. If the pupil is small and is not readily dilated with atropin, an iridectomy may first be necessary. In so-called *disk-shaped cataract*, that is, where an anterior polar one is set in a ring of clear or partially clear lens substance, an attempt may be made to dislocate the disk with a needle and let it fall to the bottom of the anterior chamber. Discission is usually employed in the case of both complete and partial (central zonular) congenital cataracts. The discission may be repeated at suitable intervals until absorption is complete, or a linear extraction may be performed shortly after the first needle operation, or Ziegler's method may be employed (page 758). It has been recommended to attempt to improve the vision in some cases of partial congenital cataract by an optical iridectomy. Pyramidal, punctate, and fusiform cataracts are not generally amenable to operative treatment. Discission or one of its substitutes is the method of operating in cases of *after-cataract*. Sometimes an opacity of the hyaloid membranes is noticeable after cataract extraction, which Fink calls *hyaloid cataract*, and Ziegler has described an *adventitious hyaloid membrane* after cataract extraction or in any case of aphakia where the vitreous has been exposed to the action of the aqueous.

Extraction of *unilateral cataract* usually does not improve the patient's visual acuteness because, owing to the inequality of refraction, the eyes will not work together. The operation may be performed (simple extraction) for cosmetic reasons, and should be performed to avoid overmaturity in the opaque lens, and to improve the field of vision upon the affected side. If there is divergence, a subsequent

tenotomy of the externus or advancement of the internus may be necessary.

The technic of performing the various methods of cataract extraction, the dangers and accidents, is described on pages 760–779.

After a successful extraction or solution, and after sufficient time has elapsed to secure firm healing, a suitable pair of lenses should be adjusted—one for distant vision and one for reading.

Removal of the crystalline lens produces the condition technically spoken of as *aphakia*, and causes a high degree of hyperopia, in the emmetropic eye corresponding to about 11 D. The degree of hyperopia will be diminished if the previous refraction has been myopic, and it is possible to produce emmetropia provided the former nearsightedness has been of such degree that the removal of the lens exactly neutralizes it.

In ordinary circumstances the correcting lens for distant vision is about +10 D. For reading and similar occupation a lens having a focal distance of 25 to 33 cm. is added to the distance glass.

In addition to the hyperopic refraction which follows cataract extraction, a certain amount of regular astigmatism is the result of the operation, due probably to failure of the wound to heal evenly on account of inaccurate coaptation of its edges, caused by the character of the incision or by some condition—for example, badly applied dressings—during convalescence. This astigmatism is generally "inverse," and is often higher during the first month or two after the extraction, or until cicatrization is complete, than it is at a later period. Usually not more than 3 D remain permanently, but even 1 D should be sought out and corrected. Naturally, prolapse or incarceration of the iris causes a very high degree of astigmatism.

Glasses should not be adjusted until all redness has disappeared from the eye, and they should not be worn constantly at first. It is wise to wait from six weeks to two months before ordering the glasses for constant use.

The amount of vision obtained after a cataract extraction varies considerably. Perfect vision is frequently secured—*i. e.*, $\frac{6}{6}\left(\frac{20}{xx}\right)$, but often patients must be content with lower degrees, $\frac{1}{8}$ or, according to some operators, $\frac{1}{10}$ of normal vision being considered sufficient to place the case within the category of successes, but vision of this grade does not constitute a satisfactory success even if it be conceded that it is an operative success.

Acuteness of vision may be considerably raised often to the normal standard by successfully dividing the capsule of the lens which remains behind, and some surgeons perform this operation almost as the rule (see Operations). One of the advantages of successful intracapsular operations is that secondary operations are not required. Removal of a central piece of the anterior capsule with capsule forceps usually obviates the necessity of secondary operations.

Apparent *accommodation in aphakia* has been noted. The various theories offered in explanation of this phenomenon have been summarized by Zentmayer: increase in the index of the refractive media; partial regeneration of the lens; eliminating the circles of diffusion by contraction of the pupil, a small opening in the after-cataract or contraction of the fissure of the lids; and forward bulging of the anterior surface of the vitreous. The adjustment of the correcting lens for distance may explain some of the cases; in others elimination of the circles of diffusion, as noted above, would seem to account for the condition.

Dislocation of the Crystalline Lens.—This may be congenital (*ectopia lentis*), and is due to a relaxation or absence of the zonula. The displacement ordinarily is *incomplete*, and really consists in a decentration of the lens; but *complete* congenital luxation is also described. Generally the displacement is lateral, upward, or upward and outward,

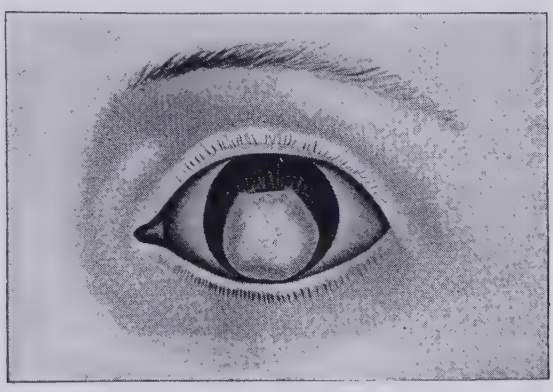


Fig. 216.—Spontaneous dislocation of lens into the anterior chamber of highly myopic eye (from a patient in the Philadelphia General Hospital). Drawing by Dr. Randall.

but in the course of time the lens may leave this position, owing to elongation of the zonular fibers, and be displaced downward and outward. Several members of the same family may be affected; for example, G. Griffin Lewis has reported *hereditary ectopia lentis* extending through six generations and involving 16 individuals. Congenital dislocation of the lens has been described.

In addition to congenital dislocations, there are those due to disease of the eye—*e. g.*, choroiditis, malignant myopia, etc.—and those caused by traumatism. Traumatic dislocation may also be *incomplete* or *complete;* if the latter, the lens may be dislodged from its normal position backward into the vitreous, forward into the anterior chamber, or, through a wound, beneath the conjunctiva, and even under Tenon's capsule.

Symptoms.—If the dislocation is partial, the margin of the lens may be seen as a dark line with the ophthalmoscope, the refraction of

the eye varies accordingly as the patient looks through the displaced lens or beyond it. The iris is tremulous from loosening of the suspensory ligament and lack of the support of the lens (*iridodonesis*), and monocular diplopia and impaired or absent power of accommodation are demonstrable. If there is complete posterior luxation, the symptoms are much the same as where the lens has been removed by opera-



FIG. 217.—Congenital dislocation of the crystalline lenses, up and out (patient in the University Hospital).

tion, and if the cause of the dislocation is trauma, the symptoms of the injury—*e. g.*, hemorrhage, etc.—may be present.

A dislocated lens usually becomes cataractous, and often causes intense pain and frequent attacks of iritis, or, by occluding the angle of the anterior chamber, may give rise to glaucoma.

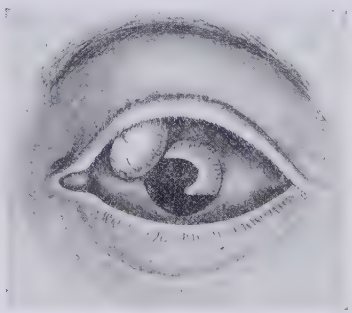


FIG. 218.—Subconjunctival dislocation of the lens (from a patient in the Chester County Hospital).

Treatment.—In partial dislocation an attempt should be made to secure the best vision with suitable glasses. Sometimes it is possible to remove an incompletely congenitally luxated lens by linear extraction following a discission, or to extract with the aid of a loupe.

In complete luxation into the anterior chamber the lens may be removed by a simple corneal incision. For removal of a lens dislocated

into the vitreous humor, provided it is producing irritation, a scoop introduced through a peripheral corneal incision may be employed, or the operation devised by the late C. R. Agnew may be attempted, namely, a double needle or "bident" is thrust into the vitreous humor far enough back to avoid wounding the iris, the handle of the instrument is depressed, the lens is caught and brought forward through the pupil into the anterior chamber, and removed in the ordinary way. H. Knapp preferred, in these circumstances, after thorough local anesthesia, to expel the lens by methodical external pressure through an upper corneal section, after removal of the speculum. He pressed the edge of the under lid on the lower part of the sclera, directly toward the center of the eyeball. If this failed, he introduced a wire or metal spoon through the corneal section and the pupil, and extracted the lens in this way. The author has employed this method with satisfaction.

If the lens has been dislocated beneath the conjunctiva, it should be extracted through a small incision made directly over it.

After the successful removal of a dislocated lens the eye should be provided with cataract glasses.

Foreign Bodies in the Lens.—Foreign bodies lodged in the lens usually cause general opacity. Occasionally the body is surrounded by a small opacity which remains localized, the remainder of the lens being clear. If a piece of steel or iron is embedded in the superficial layers, it may be dislodged by means of an electromagnet, and even from the deeper layers by the powerful magnet of Haab. If the lens is opaque the whole crystalline lens, with the foreign body in it, should be extracted, lest the foreign body become displaced and disappear within the eye. If any difficulty is experienced in deciding the position of the foreign body, or whether a foreign body is really in an opaque lens, the *x*-ray examination should be employed. A properly prepared series of skiagrams will practically always decide the question.

Siderosis of the lens has been observed. It may develop, as Van Duyse and Danis have reported, because a small splinter of iron is retained near the lens or is embedded in it.

CHAPTER XIII

DISEASES OF THE VITREOUS

Inasmuch as the vitreous after birth contains no blood-vessels and is not subject to inflammation, it has been contended that the term *hyalitis* is not correct. Straub, however, defended its use in that he believed that irritants in the vitreous may produce chemotactic substances which attract leukocytes from the vessels of neighboring structures, and the process should be regarded as a true inflammation.

Pus in the Vitreous (*Abscess of the Vitreous*).—This condition is caused by a penetrating injury, a foreign body, or a purulent choroiditis, for instance, a metastatic choroiditis from inflammation of the cord in newborn children. It may be due to scarlet fever, erysipelas, relapsing fever, influenza, basic meningitis, cerebrospinal meningitis, etc. (see also page 398).

Purulent collections in the vitreous may be caused by an infection which passes through an operation scar from a few months to many years after apparent healing. Cystoid cicatrices are particularly dangerous in this respect. The entrance of bacteria may be facilitated by the presence of prolapsed iris tissue, thinness of the scar, a fistula, and insufficient nourishment of the cicatricial tissue. On experimental grounds a defect in Descemet's membrane appears to be necessary to permit the microbes to pass into the deeper tissue of the eye. Even where the scar is dense and there is no fistula, bacteria may enter (Dolganoff and Sokoloff).

Symptoms.—If the cornea is clear, a yellowish reflex is seen shining through the pupillary space, there are retraction of the periphery of the iris and bulging of its pupillary border. Usually one or two synechiæ are present and the tension is diminished. In addition to this, there may be a pericorneal zone of congestion connected with the inflammation of the iris and ciliary body.

If the purulent exudation in the vitreous is circumscribed, its appearance is not unlike that of glioma of the retina, and the name *pseudoglioma* has been given to this condition, especially as it is seen in children. It is, however, to be distinguished from a true glioma of the retina by the history of the case, the usual presence of the signs of iritis, the retraction of the periphery and bulging of the pupillary border of the iris, and the diminished tension of the globe. Should the process extend forward to the anterior chamber and the iris become involved in a purulent inflammation the descriptive term *septic endophthalmitis* is appropriate (Fuchs). In such circumstances it is not possible to distinguish the exudation in the vitreous.

Cases of *pseudoglioma* or *ophthalmitis* or *endophthalmitis* are especially noteworthy, as they occur in children and young subjects suffering

from meningitis. As stated, purulent inflammation of the iris may develop as a complicating factor. The retina is detached and the optic nerve inflamed. The affection has been attributed to an extension of inflammation from the meninges along the optic nerve, but Percy Flemming suggests that the meningitis and ophthalmitis are both the result of a pyemic process. The source of the pyemia may be middle-ear disease. Stephenson urges examination of the pus in the eye for the meningococcus (Diplococcus intracellularis meningitidis). This micro-organism is also responsible for some cases of purulent conjunctivitis (see also page 207). George Coats and J. Graham Forbes have carefully investigated the relation of the *Meningococcus intracellularis* to pseudoglioma, and believe that a causal relation may be established between this organism and this disease.

Treatment.—If pus has once formed in the vitreous, in the manner just described, no medicinal treatment is of avail; the ball shrinks and enucleation is usually necessary. Intra-ocular injections of chlorin-water have been recommended.

If, during the earlier stages of this affection—for instance, during the course of a low fever—the discovery is made that fine flakes of opacity are beginning to appear in the vitreous, it is possible that a vigorous supporting treatment may save the eye from destruction (Hansell).

Opacities in the Vitreous.—These are either *fixed* or *moving*, and vary considerably in shape, size, and somewhat in color. The opacities may appear in the form of membranes, bands, dots, threads, flakes, and strings; or, finally, the entire vitreous humor may give evidence of uniform loss of translucency, which on careful focusing resolves itself into a diffuse, dust-like opacity.

The fixed membranous opacities usually are adherent by two or more points to the choroid, retina, optic disk, and sometimes to the ciliary process, and even to the posterior capsule of the lens. They may exist as a membrane which crosses the vitreous and covers the optic disk, or as membranous bands running from before backward, and may be coarse, dense, and organized, or fine and more like a cobweb in texture.

Method of Detection.—The examination of the vitreous is made after the manner described on page 106.

The rapidity with which the bodies move depends upon the consistency of the vitreous humor; if this is normal, the movement is slow; if it is fluid or semifluid, the movement is correspondingly rapid.

The different layers of the vitreous may also be examined for fixed opacities by means of the upright image in the ordinary way, by first finding the optic papilla, then gradually placing stronger and stronger convex lenses behind the sight-hole of the mirror until a +16 D is in position, thus bringing everything into focus from behind forward. The observer's head must be close to the observed eye. The vitreous can also be well examined by means of the diaphragm lamp which is placed

at a distance of 40 cm. and a plane perforated mirror with a narrow hole and with a convex glass of 12 diopters behind is used. Dust-like opacities appear as a defined network (Gullstrand).

The *subjective* symptoms of vitreous opacities depend entirely upon their amount and density. There may be little or no depreciation of central vision, or this may be diminished and even entirely obliterated. Patients frequently are conscious of black and gray spots before their eyes; sometimes these assume fantastic shapes, and not infrequently these shapes repeat themselves so constantly that the patient is able accurately to describe them. The same symptoms may appear where there is no organic disease (see page 473). Alterations in the field of vision, pain, redness of the eye, or similar conditions depend largely upon associated changes, and usually are absent if the vitreous alone is affected.

Causes.—1. *Refractive error*, generally high degrees of myopia associated with changes in the choroid and posterior staphyloma.

2. *Diseases of the eye*, chiefly cyclitis, iridocyclitis, uveitis, choroiditis, and retinochoroiditis.

The shape and character of the opacities vary with the condition which has caused them. In cyclitis and iridocyclitis so-called "inflammatory opacities" are seen, in certain varieties, somewhat circular in shape, resembling large mutton-fat drops; in chronic and old-standing choroiditis flake-like or thread-like opacities are very common, especially in elderly people, and are probably due to hemorrhages having their origin in the choroid. In syphilitic choroiditis and retinitis, in addition to large, floating opacities, there may be a diffuse mist which resolves itself into the so-called *dust-like opacities* (*hyalitis punctata*), and is almost characteristic of the disease which has caused the original inflammation of the choroid and retina. These dust-like opacities are either diffused throughout the entire vitreous chamber, or are situated in its posterior layers, or anteriorly, in the neighborhood of the ciliary region.

3. *Injuries of the eye*, which have caused a hemorrhage from the choroid or ciliary region. The origin of the opacity is an extravasation of blood. In the latter case, as has already been mentioned, suppuration of the vitreous may occur.

4. *Diseased Conditions of the System, Local or General.*—Infectious diseases, wide-spread endarteritis, arteriosclerosis, gout, syphilis, tuberculosis, malaria, portal congestion, anemia, and irregular or suppressed menstruation may be responsible for vitreous opacities; also the prolonged action of arsenic.

5. *Absence of Apparent Cause.*—Opacities of various shapes, often fine and thread-like, and commonly seen in old people, occur without evident disease of the uveal tract, retina, or optic nerve. Infrequent prior to the age of fifty, they may be found at later periods of life in fully 90 per cent. of examined eyes—*senile vitreous opacities* (T. Z'Brun). Their presence is usually without serious import.

Sometimes, indeed not very infrequently, the vitreous is studded

with minute light-colored spheres; possibly a congenital condition, named *asteroid hyalitis* by Benson. The condition has been well described in this country by Stark and by Holloway. According to the latter author the opacities ("snow-ball opacities" he has named them) are globular or ellipsoid in shape, dull white and not glittering in appearance as is cholesterin. They are most frequently seen in elderly persons and are not, according to Holloway, congenital. Their composition is unknown, except that they are probably not cholesterin; it may be they contain calcium. White, glistening spots in the vitreous have also been described as evidences of *fatty degeneration.*

Prognosis.—This depends entirely upon the cause of the vitreous disease. If this has started in a purulent disease of the choroid or a purulent change in the vitreous has taken place, the prognosis is exceedingly unfavorable, and the eye goes on to destruction.

If the cause of the disease is syphilis or other constitutional condition amenable to treatment, satisfactory clearing of the vitreous may be expected; even very dense opacities disappear under proper treatment. When the opacities are due to hemorrhage, although absorption of the clot may take place, fragments and strings of fibrin remain. Both hemorrhagic opacities and others are subject to relapses.

Treatment.—In any case of vitreous opacities, provided the general condition of the eye-ground justifies it, and there is reason to believe that eye-strain may be a contributing cause, suitable lenses should be ordered, but the use of the eyes at close ranges should be discouraged.

In syphilitic vitreous disease the usual remedies are necessary. If the vitreous change is associated with an exhausted condition of the system, supportive measures are indicated.

If the patient's condition warrants it, excellent results follow sweats induced by pilocarpin, or by means of Turkish baths or an ordinary electrically lighted cabinet. Iodid of potassium and sodium are useful, as is syrup of hydriodic acid.

If the disease which implicates the vitreous depends upon constipation and portal congestion, in addition to regulated diet cholagogue laxatives should be administered. Anemia and menstrual irregularities are evident indications for treatment; in the former case the combination of bichlorid of mercury with iron is useful. If there is an active inflammatory condition, blood-letting from the temple may be practised; in fact, the treatment becomes that which is suited to the acute inflammation which has started the disorder. The use of the galvanic current has been warmly recommended by some surgeons in vitreous opacities. Elschnig has treated a certain number of eyes with vitreous opacities following hemorrhage by aspirating ½ c.c. of vitreous and replacing the loss with normal saline solution.

Muscæ volitantes (*myodesopia*) are the black specks and motes often seen in the field of vision, especially if the eye is directed toward a bright surface. They follow the movements of the eye, and are especially annoying during the act of reading, as they float across the page. They do not actually interfere with vision.

There is no true opacity of the vitreous, and the ophthalmoscope fails to detect in these instances opaque particles. They are probably due to the shadows thrown upon the retina by naturally formed elements in the vitreous bodies, perhaps the remains of embryonic tissue. Corpuscles in the retinal vessels may be seen by looking through a dark blue glass at a white cloud. They appear as small oval bodies, sometimes as strings of minute globules.

Although of no serious consequence, as far as sight is concerned, these muscæ produce an amazing amount of annoyance in nervous and sensitive persons. Patients frequently maintain that they obscure an object, floating directly in front of it, and that they assume exaggerated and fantastic shapes. They are often ascribed by the laity to disorders of digestion and torpidity of the liver, and are aggravated by the habit which their possessors form of searching for them.

Treatment.—Eye-strain should be removed by the adjustment of suitable lenses, and iodid of sodium or syrup of hydriodic acid may be administered.

Hemorrhage Into the Vitreous.—As has already been stated, many vitreous opacities result from hemorrhages from the vessels of the choroid, ciliary body, or retina. Hemorrhage into the vitreous may be due to anemia, nephritis, diabetes, arteriosclerosis, myopia, and glaucoma. According to Ridley, if the hemorrhage arises from the retinal vessels, the hyaloid is usually detached and the blood lies between this membrane and the vitreous. If the ciliary body is the source of the hemorrhage, it usually bursts through the hyaloid into the vitreous. Retinal detachment may occur, especially if the hemorrhage recurs on several occasions. Injury is a common cause of hemorrhage in the vitreous, and in such circumstances the entire chamber may be so filled with blood that it is easily detected in its natural color as a dark-red clot, sometimes being so dense that no reflex comes from the fundus.

Recurrent Hemorrhages in the Vitreous and Retina.—In certain cases, generally in young male adults, ranging in age, in Eales' series, from fourteen to twenty, but occurring also at a somewhat later age (the female sex is not immune), *spontaneous hemorrhage* into the vitreous occurs, together with hemorrhage in the retina (*recurrent retinal hemorrhage*). According to Eales such patients are "below par," are liable to constipation, irregularity of the circulation, and epistaxis. The condition has also been ascribed to congenital gout (Hutchinson), to persistent oxaluria (Leber), to indicanuria, to enterogenous auto-intoxication, to focal infections, and occasionally to syphilis. There seems no doubt that this affection may be one of the manifestations of disturbance of the functions of the endocrine organs, especially the adrenals, as Zentmayer points out in his recent discussion of this subject. The same suggestion has also been made by Fridenberg. Hemophilia and menstrual disorders, while not causative, may be contributing factors (Finnoff). The investigations of Axenfeld and Stock indicate that an important etiologic factor in recurring intra-ocular hemorrhage in

adolescence, as well as in proliferating retinal lesions and retinal periplebitis (see also page 503), is tuberculosis, the active agent being a tuberculous toxin. The important relation of tuberculosis to this affection, originally suggested by Noll and by Kipp, has been confirmed by many observers (Igersheimer, Harms, E. Jackson, Finnoff, and others). The author has made some observations with Perry Pepper on myocardial changes in the subjects of this affection. There is marked disturbance of vision depending on the density of the clot, which is likely to be imperfectly absorbed, but often the resorption of the blood is unusually rapid, only to be followed by a recurrence of the hemorrhage. To these frequently *recurring hemorrhages into the vitreous* the term "malignant" has been applied. It is possible, as Coats suggested, that anomalies in the coagulability of the blood may account for many of these cases. Finoff attributes the hemorrhages to localized pathologic weakening of the blood-vessels; the disease, according to him, is primarily retinal and early changes are found in the periphery of the retina. As the result of repeated hemorrhages vitreous membranes may form, retinal detachment may take place due to traction by scar tissue, retinitis proliferans and even glaucoma may arise (one case occurred in Eales' series). The source of the blood is probably, if not certainly, the veins of the retina, although the ciliary body and choroidal vessels have also been accused in this respect.

Treatment.—This should consist in the administration of cardiac sedatives, laxatives, and later of iodid of potassium or sodium, according to the circumstances. If arteriosclerosis is present, the usual treatment of this condition is indicated. Sometimes instead of elevated there is lowered arterial tension. The administration of calcium salts to aid the coagulability of the blood is worthy of trial; coagulose and pituitrin may be administered and thyroid extract has been successfully employed. Fibrolysin has been advocated. Injections of *hemolytic serum* have been tried, but with disastrous results (Elschnig). Intravenous injections of human blood-serum have been used (A. E. Davis) and also of horse blood-serum. Ligation of the carotid for recurring hemorrhage into the vitreous has been performed, and in a few instances, it is said, with success. As in other vitreous changes, if the general condition permits it, a sweat cure may be instituted either by means of the Turkish bath or pilocarpin; active diuresis is advisable. Enterogenous auto-intoxication should be corrected as well as all other sources of focal infection. Because of the relation of tuberculosis to this condition the advantages of tuberculin treatment should be employed.

Synchysis (*Fluidity of the Vitreous*).—This is a softened or fluid condition of the vitreous, which, as has already been implied, can be positively diagnosticated or, rather, assumed to be present only by noticing the rapid movement of opacities contained within it during motions of the eye. Although tremulousness of the iris is sometimes seen where there is decided fluidity of the vitreous humor, this symptom does not prove its presence, but only a lack of support by the crys-

talline lens owing to relaxation of the zonula. The tension of the eyeball may be diminished (*hypotony*).

It occurs in elderly people with disease of the choroidal coat and in high myopia. A fluid vitreous is a serious complication in cataract extraction.

Synchysis scintillans is a term applied to a fluid vitreous which holds in suspension numerous scales of cholesterin which move with great rapidity across the ophthalmoscopic field and produce a striking picture, resembling a shower of brilliant crystals. Poncet has reported in this connection tyrosin and crystallized phosphates, but investigations seem to show that the appearance is due solely to cholesterin.

The affection probably depends upon a choroiditis, is essentially a condition of advanced years, and is said to be more common among alcoholic subjects and those with arthritic tendency or any serious



Fig. 219.—New blood-vessel formation in the vitreous of a syphilitic patient.

disorder of nutrition; syphilis, arteriosclerosis, albuminuria, and diabetes have been noted as possible etiologic factors; some cases appear to have a traumatic origin (Roemer). The affection is, however, clinically at least, seen in eyes which apparently are not diseased in other respects, especially in old people, and may be present in advanced degree without depreciation of visual acuteness. (See also page 473.)

Treatment.—This does not appear to have any influence. Succinate of iron has been recommended.

Blood-vessel Formation in the Vitreous.—Occasionally cases are examined which present an entirely new blood-vessel formation in the vitreous in front of the entrance of the optic nerve (Fig. 219).

Only a few vessels may be present or, in extreme cases, the entire disk is obscured by a congeries of contorted vessels, the whole forming an extensive vascular veil of anastomosing capillaries coming directly

from the nerve-head and having no connection with the retinal vessels. The vessels may owe their origin to vitreous and to retinal hemorrhages; in other cases syphilis is the etiologic factor. The relation of tuberculosis to this and similar conditions has been described. (See also pages 502 and 503.)

Foreign Bodies in the Vitreous.—These are usually chips of steel, splinters of glass, particles of gun-cap, or small shot or fragments of shrapnel. They may reach the vitreous by penetrating the sclera directly or by passing through the cornea and lens. The foreign body may convey infection into the injured eye; sympathetic ophthalmitis may develop. The symptoms, diagnosis, and treatment of foreign bodies in the vitreous have been included with injuries of the sclera on pages 328–332.



FIG. 220.—Cysticercus of the vitreous (from a patient at Fort Ogelthorpe, Ga.).

Entozoa in the Vitreous.—Three different species of tapeworm larvæ are known to occur in the eye and its adnexa, namely, those of *Tænia solium*, *Tænia echinococcus*, and the *bothriocephalid* tapeworms. Of these, *Cysticercus cellulosæ* is the most common.

Among 807 observations on cysticercus tabulated by Vosgien, 372 were concerned with the eye, and of these, 120 of the retina and 112 of the vitreous (quoted from Ward). None the less, in individual experiences ocular cysticercus is rare.

The intra-ocular situation of the parasite may be in the anterior chamber, in the posterior chamber, even in the lens, in the posterior segment of the eye, that is, under the retina, and in the vitreous.

Most frequently the parasite has been found in the retina, having gained entrance into the vessels of the choroid, and from there passed beneath the retina, which it detaches from the choroid. The appearances are those of a bluish-gray bladder, with a margin of lighter color, usually under a circumscribed retinal detachment; spontaneous movements are sometimes visible. The parasite, however, is able to develop floating in the vitreous (von Graefe, Fuchs), having found its way into a vessel of the retina or ciliary body. If the parasite is free in the vitreous the appearances may be as they are depicted in the accompanying diagram from a case studied by the author and Meyer Wiener. Distinct peristaltic motions and the movements of the hard neck and body were visible. Usually a progressive iridocyclitis develops. An endeavor to remove the parasite through a scleral incision should be made.

At one time this condition was frequent in northern Germany, but even here, owing to the improvement in meat inspection, etc., there has been a noticeable diminution of this form of tapeworm infection. So far as the author is aware, *Tænia solium* has not been found in native Americans, but only in those who have emigrated to this country, and even so, it is very rare among them.

Another parasite which has been seen in the vitreous, the removal of at least one specimen being on record, is the *Filaria sanguinis hominis.*

Detachment of the vitreous, except in the region of the ciliary body where separation does not occur, is produced by an accumulation of fluid between it and the retina or between it and the lens.

Traumatism, choroiditis, hemorrhages, intra-ocular growths, and staphyloma may cause it. The vitreous humor is said to be occasionally detached without change in its translucency, although opacities are usually present. Shrinking of the vitreous after a blow on the eye causes its hyaloid to be detached from the retina. In eyes removed after injury, stretching across the globe behind the lens, the so-called *cyclitic membrane* may be seen. Elschnig maintains that many so-called vitreous detachments depend upon artifacts the result of the methods used in preparing the specimens. But Sir William Lister's elaborate studies have demonstrated that detachment of the vitreous is common during life and its presence in pathologic specimens is not necessarily due to fixing reagents, especially if formalin is used; that if the vitreous from any cause (hemorrhage, injury, inflammation) adheres to the retina at certain spots and traction is exerted on the retina, it may be detached, folded, or torn (*holes in the retina*); and that holes in the hyaloid and wrinkling of this membrane may occur. Following vitreous detachment punctate deposits on the retina may appear; also fan-shaped fibrous tissue extending from the optic disk.

Hernia of the Vitreous.—This is a sac-like or globular protrusion of the vitreous into the anterior chamber, and has been noted after dislocation of the lens, after cataract extraction, and after absorption of the lens following trauma, and after contusion injury associated with laceration of the iris sphincter (Vogt). It is probably not very uncom-

mon, especially after intracapsular extractions of cataract, as some slit-lamp studies seem to indicate.

Persistent Hyaloid Artery.—During fetal life the vitreous humor is traversed by the *hyaloid artery*, which is an extension of the central artery of the retina, and proceeds from the optic nerve to the posterior surface of the lens. The vessel passes through a channel having a delicate membranous lining, known as the *canal of Cloquet*. Obliteration of this artery begins at the end of the fifth month of gestation.

Sometimes obliteration fails, and the most important congenital anomaly of the vitreous is evident—namely, the persistence of some vestige of the hyaloid artery. It may appear in the following forms:

A rudimentary strand attached to the disk; a strand attached to the disk and a vestige also at the posterior surface of the lens; a strand passing from the disk to the lens; a similar strand containing blood; a strand attached to the lens alone; and a persistent canal (canal of Cloquet) without any remnant of the vessel. These are the most ordinary and well-recognized forms.

In addition to this, shreds of tissue and membranes on the optic disk, masses resembling connective tissue, and small cystic bodies are probably remnants of this artery. Its rôle in producing posterior capsular cataract has already been described. The appearances are readily recognized by the ophthalmoscope, and require no further description than the names already given.

This classification has been condensed from the admirable monograph of Dr. De Beck who has written a complete account of the anomaly. According to Uribe Troncoso, a free cyst developed from the ciliary processes may give rise to the appearance of a vesicle floating in the vitreous. A notch in the lower part of the vitreous has been described (*coloboma of the vitreous*).

CHAPTER XIV

DISEASES OF THE RETINA

Hyperemia of the Retina.—Although the capillary network of the retina, invisible in ordinary circumstances, may, under other conditions, become evident (*capillary congestion*), the presence of a congestion is inferred, not by any alteration in the appearance of the retina itself, but by changes in the surface of the optic disk, generally described as *increased redness* or *undue capillarity*, and is associated with increase in the amount of the retinal striation which surrounds the papilla, so that its edges are veiled or slightly blurred. Such appearances are common in ametropic eyes, and in persons whose occupations expose them to the glare of artificial heat—*e. g.*, puddlers.

It is possible to speak with more confidence of a change in the caliber, course, color, and general size of the retinal vessels, provided more than the normal amount of blood finds its way into them and they are distended, tortuous, or positively lengthened. It is customary to describe the hyperemia as *active* if an increased amount of blood is sent to the retina, because the systemic circulation is unduly filled—*e. g.*, in rapid action of the heart in fever, pneumonia, etc.—and as *passive* if the blood is not properly returned from the eye, for example, in compression of the retinal vein. In the last-named circumstance the veins are large, filled with dark blood, and often tortuous, while the arteries are unaffected or are smaller than usual.

Among the *general causes* of a stasis-hyperemia may be mentioned mitral disease, emphysema, violent cough, convulsive seizures, or, in short, any cause which is likely to produce engorgement of the veins of the head and neck, and to prevent the emptying of their contents into the great venous channels of the chest. Increase in the diameter of the veins is much more frequent than increase in the diameter of the arteries, while, on the other hand, increase in the diameter of the arteries is uncommon as compared with a diminution of their caliber. Pathologic significance must not always be ascribed to apparent changes in the diameter of the veins, because eye-grounds are often crossed by large dark veins, the arteries being small by contrast, without definite local or general cause for the phenomenon.

Ordinarily patients with hyperemia of the retina do not present characteristic symptoms, but if the condition is connected with ametropia there are ocular pain, photophobia, and lack of eye endurance.

Treatment.—In hyperemia dependent upon errors of refraction the evident treatment is physiologic rest under the influence of atropin, and later suitable correcting glasses. If the condition depends upon general causes, these furnish the indications for treatment.

Anemia of the retina is not a clinical entity, but a symptom of local pressure or of some cause situated within the general economy.

The highest type of anemia of the retinal vessels is caused by stoppage of the circulation by an embolus or thrombus, and occurs in marked degree as the result of compression, in consecutive atrophy of the optic nerve. Other causes of anemia of the retina are general anemia, cerebral anemia, and syncope.

Extreme narrowing of the retinal arteries is occasionally seen as the result of a vasomotor spasm—for example, in "sick headaches" and in true migraine. In these cases there may be temporary complete or partial (hemianopic) blindness. If the blindness approaches from above downward, the obstruction is in the retinal circulation, but if it assumes a lateral form, the cortical visual centers are probably affected (Priestley Smith). Impeded retinal circulation may be attributed to the high arterial tension which is known to be present in some cases of migraine.

Under the name *ischemia of the retina* a condition is described in which, with complete blindness, there are pallor of the optic disks and extreme narrowing of the retinal blood-vessels. This phenomenon is visible, for example, in the collapse stage of cholera (Graefe), in whooping-cough (Knapp, Noyes), in erysipelas (Ayres), and under the influence of toxic doses of quinin, ethylhydrocuprein, and salicylic acid.

Treatment.—The flagging circulation should be stimulated by digitalis and strychnin. Nitrite of amyl has been employed in spasm of the retinal arteries. General anemia should be corrected by appropriate remedies.

Hyperesthesia of the Retina.—This is characterized chiefly by the *symptoms* which indicate a supersensitive state of the retina—dread of light, lacrimation, blepharospasm, neuralgic pain, and imperfect eye endurance.

Ophthalmoscopic changes may be practically absent, but in most instances those lesions will be detected which have been referred to under *congestion*, but which, adopting a name which was originally employed by Jaeger and later used by Loring, may be described as *irritation of the retina*. These are: undue redness of the nerve-head, veiling of its nasal edges, from which, and from those above and below, distinct striation of the retinal fibers are evident, while streaks of light tissue can be followed along the course of the larger vessels. The margins of the disk are veiled by this retinal striation, and although the physiologic cup, or the "light-spot," may be unchanged, the general surface of the disk seems to be covered with a delicate layer of edematous tissue. At the same time the choroid reveals changes similar to those described on page 385, or else is distinctly granular and macerated. Often the entire fundus fails to present a distinct ophthalmoscopic picture, and may be described by saying that the details of the eye-ground are not sharply seen with the aid of any correcting glass.

Causes.—Hyperesthesia and irritation of the retina are usually caused by errors of refraction and anomalies of muscle balance, espe-

cially in neurasthenic and hysteric subjects. They also owe their origin to chronic headache, neuralgia, sexual abuses, prolonged fevers, pulmonary disorders, and exposure to bright light. In a series of cases which the author has reported, oxaluria appeared to be the source of trouble.

In some instances of retinal irritation the cause seems to be dependent upon changes in the nasopharynx; for example, engorgement of the septum, associated with myxomatous and hypersensitive spots, vasoparetic and infiltrated turbinals, and secondary changes in the pharynx and larynx. Retinal irritation may sometimes be the forerunner of organic change in the optic nerve (Loring).

Treatment.—Spectacles are not a panacea, and although errors of refraction should always be neutralized, correcting lenses alone do not suffice to relieve the symptoms. General tonics, rest, massage, and all measures calculated to overcome debility or existing neurosis are required. The nasopharynx should be explored. A thorough examination of all organs should be instituted and treatment directed according to the findings. Retinal irritation is apt to be exceedingly stubborn.

Anesthesia of the retina (*neurasthenic* or *nervous asthenopia*), like several other disorders of the retina just considered, should be regarded not as an affection peculiar to the eye, but as one of the symptoms of a complicated neurosis.

The *subjective symptoms* of this condition have been arranged by Wilbrand and others as follows: Headache, particularly throbbing in the brow and temples, occipital distress, pain in the back of the neck and spine, vertigo, muscæ volitantes, defective accommodation, intolerance of light, and improvement in vision in the dusk and by wearing tinted glasses. Any attempt at concentrated vision is followed by a rapid disappearance from view of the object which the patient attempts to regard. There are diminution of central vision, sudden attacks of obscuration of vision, processions of scotomas, visual hallucinations, lack of fixation of the optical memory images, persistent and confusing after-images, colored vision—for example, erythropsia—and a red appearance of the pages of a book, the letters of which seem to be green.

In this affection peculiar alterations of the visual field, the so-called fatigue contractions, appear. The following forms have been described: The *shifting* or *displacement type*, originally investigated by Förster, in which the visual fields differ according as the examination is conducted from the temporal side to the nasal, or from the nasal side to the temporal, the contraction being pronounced on the nasal side in the former, and on the temporal side in the latter; the *exhaustion type* of Wilbrand, in which the test-object is moved from the temporal side to the nasal, and from the nasal side to the temporal, several times in succession, across the entire width of the perimeter—indeed, as often as the field continues to diminish; *unstable concentric limitation*—that is, a field which is constantly changing during examination; the *exhaustion-spiral type*, in which the tracing of the visual field appears

coiled like a watch-spring, in consequence of its limits becoming concentrically smaller; and, finally, the *recuperation-extension type*, in which the restricted field may extend during rest or by a strong effort of will (see also page 582).

The "oscillating field" described by Wilbrand and O. Koenig may also be found, in which the object disappears and reappears several times in the same meridian, and in which a similar oscillation occurs when colored test-objects are employed. Such fields are not only encountered in so-called nervous asthenopia, but in the retinal exhaustion which is found in a variety of conditions.

Patients thus affected are, for the most part, women, often the subjects of ovarian and uterine diseases, neurasthenia, hysteria, and chlorosis. It is not an uncommon affection in children between the ages of nine and fifteen, in whom, in addition to reduction of central visual acuteness, there is marked contraction of the visual field. Pure types of retinal asthenopia are also seen in men.

Treatment.—This should include all suitable general measures, and not infrequently a "rest-cure," namely, rest with seclusion, forced feeding, massage, and electricity.

Although tinted glasses are recommended, they are not always advisable, lest the affected eyes become too much accustomed to the dull light afforded through such protection. Any error of refraction should be corrected, but spectacles and treatment designed to relieve ametropia and imbalance of the ocular muscles are usually not alone sufficient to cure these patients. It should be remembered, however, that errors of refraction are often the source of the trouble, and that they must always be thoroughly and carefully corrected if good results are to be obtained. The neglect of this part of the treatment has been the origin of many cases of chronic invalidism.

Cyanosis of the Retina.—This name is applied to an ophthalmoscopic picture seen in patients with congenital heart disease and general cyanosis. The vessels of the fundus are dilated, especially the veins, which may be greatly distended and tortuous. They carry blood much darker than is normal, and the arteries resemble in color the ordinary retinal veins. Small hemorrhages near the disk and larger ones in the macula may be present; sometimes vision is normal; sometimes it is greatly reduced. The ophthalmoscopic appearances of the affection were first described by H. Knapp; they have been studied and depicted in this country by Posey, H. H. Tyson, and T. B. Holloway. In *cyanotic polycythemia* the veins of the fundus are greatly enlarged and are very dark colored; the arteries are not materially changed. Retinal hemorrhages may be present (E. Jackson, W. R. Parker).

Retinitis.—Under the general term *retinitis* are included the various types of inflammation of the retina.

Varieties and Causes.—Retinitis, like iritis and choroiditis, may depend upon constitutional disorders, altered states of the blood and blood-vessels, infections, auto-intoxication, toxins and traumatisms

or be due to an extension of a diseased process from an inflamed iris, ciliary body, or choroid—that is, the retinitis is either *primary* or *secondary*. Retinitis is often classified according to the probable etiology—for example, *syphilitic*, *renal*, *diabetic*, *hemorrhagic*, etc., retinitis. It is further divided, according to its character, into *circumscribed* and *diffuse*, and was formerly separated, according to its supposed pathologic nature, into *serous* and *parenchymatous* retinitis.

Pathologic Anatomy.—In the acute stage of retinitis the retina exhibits edema and infiltration with leukocytes and red blood-corpuscles. White areas are visible, due to fatty degeneration of both nervous and supporting tissues, varicosity and swelling of the nerve-fibers, and to masses of fibrinous exudation in the granular and nuclear layers. The blood-vessels are thickened, often obliterated, and the supporting tissue hypertrophied. In the later stages of atrophy the retina consists of a connective-tissue network which contains many pigment cells; the nervous elements disappear, and the blood-vessels are converted into solid cords. In brief, as Ginsberg summarizes the matter, the changes which present themselves for consideration include edema and exudation, hemorrhage, and small-celled infiltration; proliferation of the neuroglia and the vessel wall connective tissue; degeneration of the retinal elements, the vessels, and the neurogliar tissue; and pigmentation.

Symptoms.—Certain *objective* and *subjective* symptoms are present in most of the forms of retinitis.

1. *Loss in the Transparency of the Retina.*—This may manifest itself as a faint, diffuse haze, a circumscribed opacity and swelling, or as streaks of white infiltration, especially along the lines of the larger vessels.

2. *Areas of Exudation.*—These are an advanced stage of the condition just described. They appear as white spots, sometimes discrete, sometimes confluent, or as patches of bluish-gray, buff, or yellowish color. They should be differentiated from the shining white plaques due to atrophy of the choroid by their softer tone, their situation, and because there is an absence of accumulation of choroidal pigment. They may be present anywhere in the retina or localized in the macular region.

3. *Tortuosity of the Vessels and Change in Their Caliber.*—The veins are darker than normal, unduly wavy in outline, or positively lengthened in their course. The arteries may not be materially changed, but the finer transverse branches are often very tortuous, and both sets of vessels are liable to displacement from their normal level as they cross areas of thickening, or to partial obscuration by the puffy and infiltrated retina. Many vessels invisible in health become injected in retinitis and form a fine red striation, passing from the nerve-head. Pulsation of the vessels is readily induced by pressure.

4. *Hemorrhages.*—These occur either in the fiber-layer or the deeper portions of the retina. The presence of retinal hemorrhage alone, however, does not indicate the existence of inflammation, as it may occur quite independently of retinitis.

If the hemorrhage is in the nerve-fiber layer, it usually assumes a *flame shape*, with frayed or feathery edges; if its situation is in the deeper layers, it has a cleaner-cut border and more rounded shape.

5. *Changes in the Nerve-head.*—More or less change in the optic papilla is present: undue redness, loss of the distinctness of its margins, obscuration by the swollen and puffy retinal fibers, or, finally, positive inflammation or neuritis. Atrophy of the disk is commonly present after severe retinitis.

6. *Pigmentation.*—Black spots of pigment mark the situation of former retinal hemorrhages. Pigment in the retina, like hemorrhages, although in many instances a sequence of retinitis, is of itself not necessarily a symptom of inflammation of this membrane.

The difference between pigment in the retina and in the choroid has been described on page 386.

7. *Atrophy of the Retina.*—This, like atrophy of the choroid, may indicate a former hemorrhage or an area of inflammation. All the retinal layers, as well as the choroid, may be involved, exposing a white patch of sclera (*atrophic choroidoretinitis*), or only the superficial layers may be affected, and the spot may be marked by a permanent whitish or yellowish opacity. Contraction of the vessels and white tissue along their coats are often seen after retinitis.

In addition to the ophthalmoscopic signs there are:

1. *Change in Visual Acuteness.*—Central vision is *diminished* in direct proportion to the severity of the case and the situation of the inflammatory action.

2. *Change in the Field of Vision.*—This may be irregularly or concentrically contracted, or scotomas may appear in its center.

3. *Distortion of Vision.*—This occurs under several forms: (*a*) Objects appear to be reduced in size if the retinal elements are spread apart (*micropsia*); (*b*) objects appear to be increased in size if the retinal elements are crowded together (*macropsia*); (*c*) objects appear to undergo change in their contour or shape (*metamorphopsia*). Vertically placed parallel lines, on the one hand, appear to be bulged outward, and, on the other, to be bent inward. Fine parallel lines may appear *wavy* to a normal eye. Retinal metamorphopsia is often associated with a scotoma.

4. *Pain and Photophobia.*—Acute pain is almost always absent, even in violent forms of retinal inflammation; indeed, it is much more likely to be present in the less pronounced grades.

Usually the sensation is one of discomfort rather than of actual pain. Photophobia may or may not be present. It is never a marked sign, although comfort ensues from the use of tinted glasses.

Diagnosis.—This depends upon the essential symptom of the disease—opacity or loss of transparency in the retina. All the other symptoms which may be present—exudation, hemorrhages, pigmentation, and atrophy—help to make up the clinical characteristics of the various types, but in themselves are not diagnostic of inflammation of this membrane.

Much diagnostic aid is obtained by noting the effect of the disease upon vision, especially under the influence of diminished illumination, and if acuteness of sight fails quite out of proportion to the amount of the light reduction, the student should at once be upon his guard. Investigation of the light-sense in the manner already described (see page 70) is important. If the coarse changes detailed in the general symptom-grouping are present, the picture is readily interpreted.

Course and Complications.—The course of a retinitis, like any other inflammation, may be *acute* or *chronic*, and its progress of long or short duration. When the retina and choroid are simultaneously inflamed, a common complication is change in the vitreous (*vitreous opacities*), and an almost constant association is inflammation of the optic papilla, followed by atrophy (*retinitic atrophy*).

Prognosis.—This may be favorable, grave, or positively fatal, depending upon the extent of the inflammation, its situation in the inner or outer layers of the retina, and the cause. Before giving a prognosis the surgeon must always attempt to estimate the extent of the permanent disability which is likely to remain in the form of atrophy of the membrane or secondary changes in the papilla. Other things being equal, the prognosis of syphilitic retinitis is the most favorable.

Treatment.—This, in general terms, demands perfect rest for the inflamed organ, and therefore atropin mydriasis is often desirable. In sthenic cases, in the early stages, blood-letting from the temple has been recommended.

The remedies most likely to afford relief are the various forms of mercury, iodid and bromid of potassium, pilocarpin, and electric-cabinet diaphoresis and Turkish baths. Special methods of treatment are reserved for the sections devoted to the several clinical varieties.

Types of Retinitis.—As an introduction to the special varieties of retinitis which will presently be considered, it serves a useful clinical purpose to refer to two types of retinitis formerly described under the names *serous* and *parenchymatous retinitis*. The first type, also called *retinitis simplex*, *diffuse retinitis*, and *edema of the retina*, is a condition characterized by an infiltration, especially of the nerve-fiber and ganglionic layer of the retina, causing opacity, together with hyperemia, most marked in the veins.

The opacity varies from a delicate veiling to a decided grayish-white opacity, most noticeable around the nerve-head, the margins of which are veiled or hidden. From this point the grayish opacity shades out into the surrounding retina. The disk is not necessarily swollen: it may be simply hidden by the infiltrated tissue, or, if this is not marked, it is very red and its edges obscured by the radiation of finely injected capillaries from its margins. The veins are dark, fuller than normal, tortuous, and often partly covered by the swollen tissue; the arteries are not much changed in size, unless perchance they may be reduced in caliber by compression. Hemorrhages are rare, and exudations in the macular region are uncommon.

There are no external signs of this form of inflammation. Both

direct and indirect vision are affected, the former being "foggy," the latter concentrically contracted.

The second type, also called *deep retinitis,* includes those forms of retinitis in which, in addition to edematous infiltration, opacity of the retina, and venous hyperemia, there are pronounced cellular infiltration and structural change, ending finally in atrophy of the elements.

Exudations of yellowish or gray color are visible, occurring in patches throughout the eye-ground, and often localized in a characteristic manner in the macula. Small hemorrhages are commonly present, and the morbid processes may attack the sheaths of the vessels, causing thickening and hypertrophy.

There are no diagnostic exterior ocular manifestations. Deeply seated pain of a dull, aching character may be present. Vision is often much disturbed, varying from a mere fogginess of the outlines of objects to an almost absolute loss of sight. Contraction of the field of vision and positive scotomas are demonstrable, and the phenomena of distortion of objects are apparent. The disease may be circumscribed or diffuse, and localized in the external or internal layers, or affect both of these and also involve the choroid.

The **prognosis** of the second variety is always grave, and although in certain cases absorption of the products is possible, compression and atrophy of the nervous elements must result in most instances. Independently of the fact that so-called serous retinitis may be the initial change of other forms presently to be described, it has been ascribed to cold, to undue light and heat, to toxins, infections, and to the influence of refractive error in eyes worked under the disadvantage of imperfect illumination. The other type depends, as a rule, upon various constitutional disorders, or occurs in association with other diseases of the eye.

Partaking of the nature of one or the other of these forms there are certain clinical types:

Syphilitic Retinitis or Chorioretinitis.—The syphilitic forms of retinal inflammation have been divided by Alexander into: (1) *Choroidoretinitis;* (2) *simple syphilitic retinitis;* (3) *retinitis with exudations;* (4) *retinitis with hemorrhages;* and (5) *central relapsing retinitis.*

The *first form,* that is, *diffuse chorioretinitis,* first described by Jacobson and later by Förster, is really a disease of the choroid, and the pathologic changes of cellular infiltration, exudation, atrophy, and proliferation of the pigment epithelium are found in the choroid, between the choroid and retina, and in the adjacent retinal layers. There may be changes in the retinal vessels—that is, a *syphilitic endarteritis.* The pigment changes are produced by wandering and proliferation of the retinal pigment. The choroid is markedly altered; sometimes the choriocapillaris completely disappears. In other words, the retinitis does not depend exclusively upon a choroiditis, nor does the contrary relationship hold good.

The following signs are visible: Opacity of the vitreous, especially in the posterior portion, which resolves itself into fine points or dust-

like particles, and stretches out to the periphery like a cloud; loss of transparency of the retina surrounding the nerve-head, which may be unduly hyperemic, and on account of the fine opacity in the vitreous may give the impression that it is swollen; numerous yellowish or white spots of exudation bounded by pigment beneath the vessels of the retina in the periphery of the eye-grounds, and white spots in the macula present in fully one-third of the cases (Förster); and, finally, participation of the iris and posterior layer of the cornea which is a not infrequent complication (in one-sixth to one-eighth of the cases [Igersheimer]).

The *subjective* symptoms are: Depreciation of central vision, very marked in the later stages; markedly delayed retinal adaptation, night-

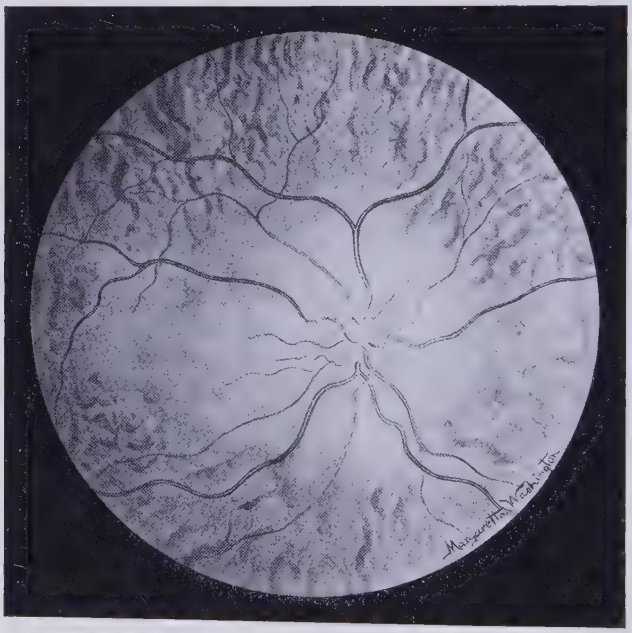


Fig. 221.—Syphilitic retinitis.

blindness and great lessening of visual acuteness under weak illumination; irregular and concentric contraction of the visual field and the formation of *ring scotomas*, sometimes complete and sometimes incomplete, as well as scotomas in the center of the field; and sector defects in the periphery and shimmerings, dancing spots and circles (photopsias), and distortion of objects in the form of micropsia and metamorphopsia due to separation of the rods and cones by the effusion.

In the *second form* there appears to be a more definite localization of the disease in the retina, particularly its inner layers, and this tissue is, as Schöbl expresses it, first selected by the syphilitic poison. The ophthalmoscope reveals a gray opacity surrounding the nerve entrance

and stretching out in lines along the vessels; the papilla is discolored, cloudy, and has been compared to a yellowish-red, oval body seen through a covering of fog. The veins are darker than normal; the arteries usually are not materially changed. Although the participation of the choroid in these processes is the rule, there is no doubt that a pure syphilitic retinitis can develop in the inner layer of the retina, independent of the choroid. Late changes which occur in syphilitic chorioretinitis and retinitis are: atrophy of the disk (*retinitic atrophy*), pigmented chorioretinitis in the form of variously shaped pigment deposits, not, however, specially disposed along the vessels, and occasionally posterior cortical cataract.



FIG. 222.—Syphilitic chorioretinitis; sclerotic arteries; cherry spot and area of edema in macula, resembling lesions caused by obstruction of central artery; small retinochoroidal lesions in periphery. (From the William F. Norris collection in the University of Pennsylvania.)

Other objective symptoms in syphilitic chorioretinal disease are floating vitreous opacities, exudations along the lines of the vessels (*retinitis with exudations, perivasculitis*), and extravasations of blood, usually round in shape, attributed to disease of the vessel walls (*endarteritis*) or to the formation of thrombi (*retinitis with hemorrhage*). Hemorrhages in syphilitic retinitis, however, are of comparatively uncommon occurrence. Preretinal hemorrhages have been observed.

According to Haab, *syphilitic endarteritis* is a comparatively rare disease, and may present the following lesions: Visible opacity of the walls of the arteries and rarely of the veins; disease of the vessel walls, manifesting itself, as in senile sclerosis, by a narrowing of the blood-

columns, and sometimes associated with extravasations of blood; an opacity corresponding to that caused by an obstruction of the central artery or one of its branches, and appearing as a grayish-white or a milky area, with ill-defined edges, in which at times considerable hemorrhage may take place; and groups of circumscribed white patches somewhat resembling those seen in nephritis.

In its late stages syphilitic chorioretinitis may be elaborate. Thus large atrophic areas edged with pigment may be evident between which are smaller disseminated pigment spots, associated with marked vascular changes, the vessels being sheathed in white lines or converted into white cords.

Date of Occurrence.—In the acquired form of the disease it appears from one to two years after infection, sometimes as early as the sixth month, and is found in about 8 per cent. of the cases (Alexander). One eye alone may be affected, but usually after several months the second eye is also involved. It is more common between the third and fourth decade of life than at other periods.

True retinitis must not be confounded with the so-called "retinal irritation" commonly seen in association with iritis, and the symptoms of which have been described (page 481). Retinitis, however, may accompany or follow iritis.

Course and Prognosis.—Although the onset of syphilitic chorioretinitis may be sudden, the course is essentially chronic.

The *prognosis* largely depends upon the stage at which treatment is begun and the vigor of the measures employed. Delayed or neglected treatment may lead to the grave consequences of extensive atrophic choroiditis, pigmentary degeneration in the retina, and atrophy of the optic disk. Even in favorable circumstances improvement may be temporary and many stubborn relapses occur. An attack of *iritis* may complicate or usher in a relapse.

Treatment.—The same constitutional measures recommended in the treatment of syphilitic iritis (see page 345) are indicated, and, in so far as mercurials are concerned, should be vigorously employed. Concerning the value of salvarsan, or arsphenamin, in syphilitic retinitis, it may be said that successes have been reported even in the presence of decided endarteritis, and in the author's experience the use of this remedy in the manner already described has been followed by admirable results. It has no evil effect on the retina. Usually a mydriatic is advisable, and in any event dark glasses may be worn.

Central relapsing retinitis (*retinitis macularis*) belongs to the late manifestations of syphilis, and appears in the form of a gray or yellow area in the macula, or as numerous small yellow or yellowish-white spots and pigment dots, or as a diffuse opacity of this region. The papilla and its surroundings are unaffected. It is a rare form of syphilitic retinitis, stubborn in its character, and prone to relapse.

Hereditary Syphilitic Choroidoretinitis.—Various types of hereditary syphilitic affections of the retina and choroid occur, and they have been particularly well described and depicted by Haab and Sidler-

Huguenin. Whether the primary seat of the disease in these cases is in the retina or in the choroid has not, in Haab's opinion, been definitely settled. According to these authors some of the following types may be encountered: (1) The periphery of the eye-ground presents a somewhat leaden color and contains black circular and triangular patches of pigment. The remainder of the fundus is thickly covered with reddish-yellow spots placed upon a dotted brownish-black surface. Occasionally these lesions are not extensive and cover only certain portions of the fundus, especially the periphery. (2) Chiefly in the periphery of the eye-ground roundish black foci of pigment, discrete and con-

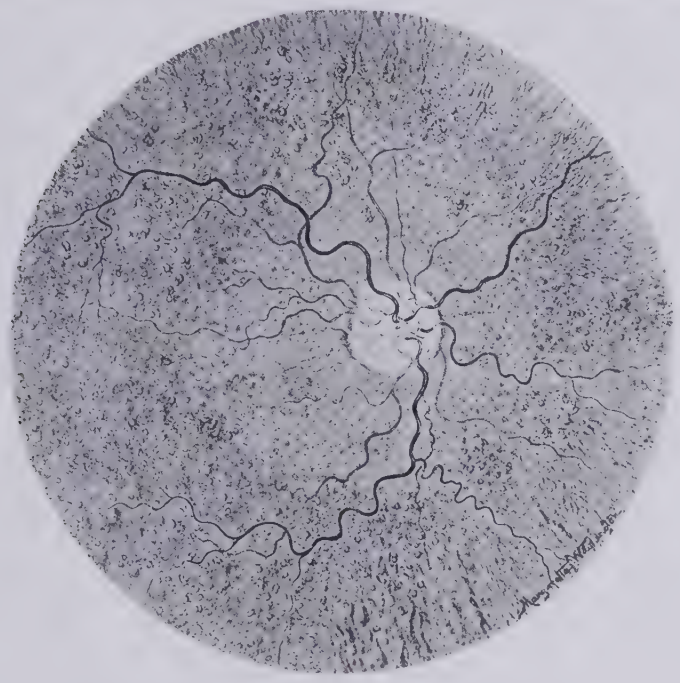


Fig. 223.—Appearances of the eye-ground in hereditary syphilis (from a patient in the University Hospital).

fluent, are evident, interspersed with linear and circular yellowish patches. The lesions are not infrequently seen after the subsidence of interstitial keratitis. (3) In place of gray and black lesions, whitish circular or confluent patches may be found in the periphery of the fundus. Sometimes these types are mixed, and in some severe cases there are coarse choroidoretinitis, diseased retinal vessels, and atrophy of the optic nerve. There may be diminution of central vision, contraction of the field of vision, and night-blindness, symptoms which are absent in mild manifestations of the disease. Other types of chorioretinitis in hereditary syphilis are: *chorioretinitis circumpapillaris*, in which for a wide space around the nerve-head are arranged small yellowish-white lesions (Igersheimer); chorioretinitis with extensive peri-

vasculitis and areas of disseminated pigmentation, clouding of the disk by an exudation, bluish white or grayish red in color which extends from it, and light-colored small lesions in the periphery (Knapp, Hirschberg). The *treatment* of the chorioretinal lesions of hereditary syphilis does not differ from that suggested in the acquired forms of the disease, except that the author cannot from his own experience testify as to the value of salvarsan or its equivalent. He has usually employed mercury and the iodids.

Metastatic Retinitis (*Septic Retinitis of Roth*).—This term has been applied to an affection especially seen in surgical pyemia and puerperal septicemia, and is characterized by small, circumscribed white spots near the papilla and in the macular region. Usually both eyes are involved and numerous small hemorrhages may be seen. These spots are due to fatty degeneration of the capillaries and infiltration of the retinal fibers, caused by the infectious emboli in the vessels. Microorganisms have been demonstrated in the lesions and in the retinal vessels.

The spread of the inflammation to the uveal tract and the relation of this condition to purulent *metastatic ophthalmitis* (choroiditis) has been described on page 399. This condition is also sometimes called *embolic panophthalmitis* or *endogenous ophthalmitis*.

An independent or *primary suppurative retinitis* may be caused by injury, that is, by a penetrating foreign body. A form of retinitis characterized by radial lines around the macula and white foci in its neighborhood, attributed to ethmoiditis, has been described (Gourfein-Welt).

Treatment.—The prognosis and treatment of suppurative retinitis does not materially differ from that recorded in connection with metastatic ophthalmitis. Occasional recoveries are recorded with preservation of eyesight.

Hemorrhagic Retinitis.—The presence of hemorrhages in the retina does not imply the coexistence of retinitis; only if signs of inflammation are added is the term "hemorrhagic retinitis" justified.

In a typical case the appearances are as follows: Swelling of the papilla, its edges being clouded or hidden by an opaque infiltration of the surrounding retina; darkly tortuous and distended veins, but small arteries; and numerous hemorrhages, linear, flame shaped, irregular, or round in shape.

The size, number, diffusion, and localization of the hemorrhages vary. Thus, they may be everywhere throughout the eye-ground, or grouped especially in the macular region or around the papilla. If white spots are present as the result of degeneration after absorption of the blood, the appearances may closely resemble those seen in so-called renal retinitis, which, indeed, may be one of the types of hemorrhagic retinitis.

Causes.—Hemorrhagic retinitis occurs with diseases of the heart and of the blood-vessels—*e. g.*, hypertrophy, aneurysm, and endarteritis; in suppressed menstruation; at the climacteric; and in a

variety of general and local diseases, sometimes presenting types presently to be described under special clinical designations. More rarely, retinitis with hemorrhages is caused by secondary syphilis.

The hemorrhages may be due to rupture of retinal vessels whose coats have become degenerated—in other words, they depend upon endarteritis; but recent investigations show that in many cases, although the arteries may be diseased, there is even more extensive change in venous coats, and there may be thrombosis of the central vein. The disease is often confined to one eye. The connection between degeneration of the blood-vessels and chronic inflammation of the inner layers of the retina is an intimate one. To all inflammations of the layers of the retina under such conditions the term *angiopathic retinitis* is applied by Wilbrand and Saenger. (For further consideration of this subject see Hemorrhages in the Retina, p. 516.)

Prognosis.—This is unfavorable because the ocular condition may indicate a grave vascular or cardiac malady, and may be the forerunner of extravasations in vital centers. Sight may be seriously impaired. Secondary changes in the retina and optic nerve are likely to follow; glaucoma frequently results.

Treatment.—The therapeutic measures must be governed by the general condition. F. R. Cross recommends subconjunctival bloodletting, and wet-cupping the temple has been advised. Often mercury, iodid of potassium, and iodid of sodium are indicated, with or without cardiac sedatives, and diaphoresis may be required.

Renal Retinitis (*Albuminuric Retinitis; Papilloretinitis; Retinitis of Bright's Disease*).—During the course of various types of disease of the kidneys changes in the retina frequently occur, chiefly characterized by edema, exudations, and hemorrhages, conspicuously evident to the ophthalmoscope, and commonly entitled *albuminuric retinitis*. For obvious reasons this term is inaccurate and *renal retinitis* is the better designation.

Symptoms.—In a typical case, beginning in the macula or its immediate neighborhood, and continuing to be most numerous in this region, variously shaped and placed white spots appear. These at first may be small, discrete, and sharply separated, but later, or under other conditions, they form a somewhat *star-shaped figure*, the rays of which surround the fovea, but for the most part do not involve it. Occasionally, instead of a stellate arrangement, the white spots and lines, somewhat radially placed like spokes in a wheel, affect this neighborhood in part, but do not completely encircle it. In the central part of the retina white patches, with soft edges, sometimes surrounded by an area of visible edema may appear: they are called "cotton-wool patches."

At some distance from the papilla, and often surrounding it, larger yellowish-white or white spots are seen, which may coalesce and form a ring-shaped zone around the nerve-head broader than its own diameter. This striking, wide white area has been compared to snow, and designated "the snowbank appearance of the retina."

Conspicuous features are the *hemorrhages*. They may be linear, flame shaped, or round, or mere flecks scattered here and there, and found with difficulty, or they constitute large, dark red extravasations. Should they disappear a whitish mark may denote their former situation. Sometimes they occur in great numbers, like fresh explosions. To a certain extent they are indications of the violence of the disease.

The blood-vessels may run over the white plaques, or may be buried in the swollen retina. Sometimes a vessel disappears beneath the infiltration, to reappear at some distance beyond. The veins are dark and often tortuous; the arteries, as in other forms of retinitis, are not materially altered in size. In the later stages the vessels exhibit lack of transparency of their walls, in the form of white tissue along the sheaths, or they are actually converted into white strings, that is, *retinal angiosclerosis* is present. Pigment dots and circles in the retina are often visible in the advanced period of renal retinitis.

Finally, the optic papilla and its immediate surroundings may be intensely hyperemic, or a swelling of the nerve-head occurs, quite indistinguishable from that of *choked disk*, as it is seen in tumor of the brain. In any circumstances the edge of the papilla is clouded, but not necessarily swollen, the surrounding retina finely clouded, and traversed by numerous radiating injected lines, like those described in other types of retinitis. Quite commonly the changes in the papilla directly join the band of infiltration already described, surrounding the end of the optic nerve.

The chief, in fact the only, *subjective* symptom is depreciation of vision, which may vary from a slight and gradual impairment to complete blindness. It is a well-known fact that Bright's disease is often discovered by an ophthalmoscopic examination, the patient being ignorant of the fact that he is the subject of serious organic malady. The visual field may be altered according to the situation of the retinal lesions, and may contain blue-blind areas. According to Gerhardt, blue-blindness may be a sign of contracted kidney, and Simon maintains that violet-blindness is not uncommon in connection with renal retinitis.

Forms of the Disease.—Although systematic writers have recognized a number of types of renal retinitis, for example, *inflammatory* or *exudative*, *degenerative*, *hemorrhagic*, and *neuritic* or *papillitic*, there is no special advantage in according them separate clinical descriptions.

Often small hemorrhages and comparatively insignificant dots in the macula may be the signs of renal retinitis, and consequently of renal disease. Indeed, the so-called *typical renal retinitis* is not so frequently encountered as the less elaborately produced lesions of this affection. Among early signs of renal retinitis are changes in the capillary circulation and dilatation and tortuosity of the small vessels around the macula, while the nerve-head assumes a congested, brick red color. In every case of retinal disease the urine should be frequently and thoroughly examined.

Causes, Date of Occurrence, and Frequency.—While in general

PLATE IV.



Albuminuric retinitis; star-shaped figure in the macula; the circulation in the distended veins impeded where the latter are crossed by the arteries which are undergoing sclerotic changes.

terms Bright's disease is the cause of the retinitis which bears its name, it most frequently occurs with chronic interstitial nephritis. It may also be caused by chronic parenchymatous nephritis, especially in the so-called inflammatory form. Naturally, the secondary contracted kidney, which is a sequence of large white kidney, may be associated with retinitis, and this is also true of amyloid disease of the kidney. Retinitis is rarely seen in acute nephritis, but it does occur, for instance, in the course of scarlatinal nephritis. During the past war an acute transitory nephritis was common; "retinitis," with edema of the retina, was a complication in many cases—the so-called *trench-nephritis-retinitis*.

Usually both eyes are involved, but *unilateral renal retinitis* is not a rarity (Knies), a certain percentage of cases, it has been stated, maintaining retinal lesions in one eye alone until death. But in a large percentage of cases the unilateral character of the affection is temporary, both eyes ultimately becoming affected. Indeed, it is probable that a strictly unilateral retinitis does not occur, although involvement of the second eye in some degree may be long delayed. In general terms it is likely that the renal disease must be present for some months before retinal lesions appear. The age at which patients are attacked is usually stated to vary from thirty to sixty, the most prolific single decade, according to Nettleship, being from fifty to sixty. It is comparatively rare before the twenty-fifth year, but children and young persons are not exempt.

About twice as many cases of renal retinitis occur in men as in women. If there is decided hyaline thickening of the retinal arteries, an early stage of granular kidney may be suspected, especially if the patient is comparatively young (Nettleship). The recorded percentage of retinitis in renal diseases varies from 9 to 33. In the author's experience fully 30 per cent. of patients with chronic Bright's disease, as he has examined them in general hospitals, have been affected by various forms of retinitis, but if these statistics should include not only the cases of so-called typical retinitis but also those of comparatively insignificant lesions, consisting chiefly of alterations in the walls of the retinal vessels and blurring of the disk, this percentage would be considerably higher.

Course, Pathologic Anatomy, and Prognosis.—Sanctioned by long usage the term "retinitis" is commonly employed to indicate the changes in the retina associated with chronic Bright's disease. But inasmuch as the evidences of active inflammation are rarely, if ever, present it may be questioned whether it should be retained. The conspicuous features are passive edema and the results of degenerative changes in the retinal and choroidal vessels.

The white spots, although they may subside, comparatively rarely disappear entirely, the macular changes being mostly permanent. "Cotton-wool patches" may disappear, at least as far as ophthalmoscopic examination is concerned, and sometimes quite rapidly (fourteen days), although at the same time others develop (Foster Moore). Discolora-

tion and atrophy of the papilla, contraction of the vessels and the formation of white tissue along their walls, and pigment changes in the retina finally result.

The *pathologic changes* are found chiefly in the macular region and in a zone surrounding the nerve. The retina is thickened by passive edema and by hypertrophy of its nervous and supporting tissue. The glistening spots and white patches, interpreting retinal exudations, are fibrinous, and later become hyaline; in some, fatty changes are notable. Their star-shaped arrangement depends upon the oblique direction of the fibers of Müller in this position, or upon tension lines in an edematous retina (Foster Moore). Many fatty granular cells and deposits of coagulated fibrin are seen, particularly in the nuclear layers, and localized and scattered pigment proliferation occurs. In many specimens disk edema is evident. Hemorrhages are present, but not necessarily a pronounced feature. In the early stages the vessels show thickening of the adventitia, and later pronounced hyaline changes and proliferation of the lining endothelium. The same causes which originate disease of the blood-vessels of the kidney also give rise to the alterations in the retinal vessels, and to these alterations the chief rôle must be ascribed in producing the various types of retinal lesions. Indeed, some authorities maintain that renal retinitis is entirely the outcome of disturbances in the circulation, that is, depends on arterio- and phlebosclerosis and their sequels. Sclerotic changes in the choroid vessel, similar to those in the retinal vessels, are also present; pigment disturbances and accumulations further emphasize the association of the choroid with renal retinitis. It is, however, difficult to explain the retinitis of acute nephritis (which occasionally occurs) and that of pregnancy, on the theory that vascular lesions are alone responsible. Hence, the influence of toxins circulating in the blood-stream cannot be disregarded. Indeed, a number of highly qualified observers insist, although unwilling to neglect entirely the etiologic significance of vascular changes in renal retinitis, upon the paramount importance of toxins in this respect. Zur Nedden believes that a material is elaborated by the decomposition of kidney substance, which toxin possesses selective affinity for the retinal tissues.

Complications.—Detachment of the retina, hemorrhage into the vitreous, embolism and thrombosis of the vessels, extravasations into the choroid, and glaucoma may be complications of this affection. Detachment of the retina is not infrequent, and glaucoma may arise exactly as it does in retinal hemorrhages and retinal angiosclerosis. According to R. Foster Moore retinal detachments in renal retinitis may be *flat*, due to solid exudation—fibrinous, granular or hyaline, or *globular*, due to accumulation of fluid. Recovery is not inconsistent with this type of retinal detachment. Retinal detachment was also noted in the retinitis or retinal edema which was frequently observed in association with *trench nephritis* during the past war (Derby, Greenwood).

Prognosis.—The prognosis, so far as vision is concerned, depends

upon the extent of the lesions and of the involvement of the macula. In general terms it is unfavorable, although fair vision is often retained. Sometimes the exudations practically disappear. In so far as the life of the patient is concerned, renal retinitis is an unfavorable symptom, and many patients die within two years after its detection, and a considerable percentage within the first year of its development. There are, however, frequent exceptions to the rule, and the records show that patients have lived five, seven, and even a greater number of years after the retinal lesions have appeared, especially if they have been detected early and suitable treatment has been instituted.

Retinitis in Pregnancy.—While the occurrence of albuminuria during pregnancy is not uncommon, varying, according to statistical reports, from 2 to 20 per cent., involvement of the optic nerve and retina, in the form of a neuroretinitis, to which the term *albuminuric retinitis of pregnancy* is usually applied, is much less frequent. The retinitis in this condition may gradually develop, occurs most frequently in primiparæ, and generally in the second half of pregnancy; exceptionally at an earlier period. The ophthalmoscopic signs of this retinitis may not differ from those which are caused by other forms of Bright's disease, and, in general terms, there is a widespread neuroretinitis with exudations and hemorrhages. The retinitis of pregnancy has been ascribed to a nephritis which is brought about by this condition, especially a fatty degeneration of the kidney epithelium, and also to toxemic products in the circulation (J. H. Fisher; see also page 496). It may also be caused by an acute nephritis which has developed during the pregnant period, and by an exacerbation of a pre-existing chronic nephritis during the same period. Retinal detachment may be a complicating condition; it may subside entirely with the disappearance of the retinal lesions.

The outcome of renal retinitis of pregnancy, however, is sometimes disastrous, atrophy of the optic nerves, thickened and sclerotic vessels, permanent macular lesions, and choroidal atrophy and pigment deposits indicating the participation of the choroid in the process. In place of retinitis the original manifestation is occasionally that of a widespread (sometimes circumscribed) *choroiditis*. After its subsidence patches of atrophy and disseminated areas of pigment deposit are evident. If they involve the macula, direct vision is greatly impaired.

In the *retinitis of pregnancy* the prognosis, in so far as it concerns the vision and the life of the patient, depends upon the duration of gestation. With the termination of pregnancy the retinal lesions may subside and good vision may be restored, provided the process has not continued so long that the secondary changes already described have taken place. For this reason the induction of premature labor has been recommended as a therapeutic measure, and if the visual disturbances appear during the first six months of gestation usually the pregnancy should be terminated if sight is to be saved.

Diagnosis.—In widespread renal retinitis the changes detailed in the symptom-grouping are striking and in a sense characteristic, but the

so-called typical cases are not as frequent as those in which the lesions are not so evident, and the significance of the retinal disease must be decided by general examination. Even so-called "typical" appearances have been observed in infections—for example, erysipelas and syphilis—independently of nephritis. Thus, Schieck has investigated a certain number of eyes with *macular changes* which were regarded as characteristic of nephritis, and yet the evidence of kidney disease was lacking; similar observations have been made by Wilbrand and Saenger, by Parsons, and by the author.

Papilledema from intracranial disease may exactly simulate this affection, and often only by a careful study of the urine and the general



Fig. 224.—Retinitis of pregnancy. (Negro patient in the University Hospital.)

symptoms the diagnosis can be established (see also page 547). The question becomes still more complicated if albuminuria is associated with brain tumor.

In glycosuria and leukemia somewhat analogous appearances are found, and again, an examination of the urine, as well as that of the blood, may be necessary before reaching a diagnosis.

The white spots are distinguished from plaques of choroidal atrophy by the absence of pigment heaping. Fine lesions of the choroid in the macular region may be mistaken for somewhat similar retinal changes; but they are more scattered, more yellow in color, usually unassociated with distinct loss of vision, and less liable to assume a stellate or radial appearance.

It is evident that a star-shaped figure in the macula is in no sense pathognomonic of renal retinitis and it may arise under various conditions and as the result of diverse factors. It may be the sole lesion or there may be an associated papillitis. Such a lesion may arise, especially in young persons, without discoverable cause, or be due to focal infections or to anemia and chlorosis. In one case reported by the author, the subject of the affection being a young woman with pronounced chlorosis, all the manifestations disappeared under the influence of iron therapy. The name *stellate retinitis* is applied to this condition.

Treatment.—Local measures are practically of no avail. The case must be managed on the general principles suited to the form of kidney disease which is present and the patient should be studied and treated in conjunction with an internist. A proper remedy in most cases is iron, usually in the form of the tincture, and often advantageously combined with bichlorid of mercury. Decapsulation of the kidney has been tried without encouraging success. Harvey Cushing has recommended cerebral decompression, because he believes that increased intracranial tension is an important factor in the development of renal retinitis. The author's single experience with this operation in these circumstances was disastrous.

Diabetic Retinitis.—This occurs in several forms. It is always bilateral, in the author's experience, but both eyes may not be affected at the same time; or in one eye the lesions are pronounced, in the other so slightly produced they may escape detection. Unilateral diabetic retinitis, however, has been described (Foster Moore). The retinitis associated with diabetes does not develop in the juvenile subjects of this disease.

Hirschberg describes two varieties of diabetic retinitis—an *exudative* and a *hemorrhagic* form. In some cases of diabetic retinitis, either with or without hemorrhage, there are widespread areas of yellowish-white exudation probably due to the coalescence of small white spots, and these lesions may arrange themselves in zone-like areas, above or below the macula, resembling the so-called circinate retinitis, or may be massed in the central region of the retina (Fig. 225). They usually are late manifestations of diabetes, and are seen at a time when gangrene, carbuncle, hemiplegia, and other serious complications of this disorder arise. In any case of diabetes of long duration retinitis is seldom absent, although it may sometimes be difficult to find the lesions, because (as before stated) they are situated in the periphery of the eye-ground. This is especially true if the complication of high myopia, or cataractous lens, is present. More commonly than in the retinitis of nephritis, opacities and hemorrhages occur in the vitreous humor, and a condition analogous to proliferating retinitis may arise.

To a collection of small white spots and hemorrhages (usually in the deeper retinal layers and not flame-shaped; sometimes scattered over the fundus in quite small circular spots) irregularly arranged in the macular region and between it and the disk the name *central punc-*

tate diabetic retinitis has been applied. By some authors this appearance is considered typical of diabetes. It may rather closely resemble arteriosclerotic retinitis (page 520).

Diabetes and chronic nephritis may be coincident and complicate the ophthalmoscopic picture; so also a diabetic subject may have generalized arteriosclerosis with the retinal lesions of that condition predominating or present to the exclusion of others supposed to be due to the metabolic disorder. It is doubted by some authorities whether a pure diabetic retinitis exists and, indeed, the evidence (recently confirmed by H. P. Wagener and R. M. Wilder in the Mayo Clinic) is strong that the metabolic disturbances are not the primary cause of the ocular

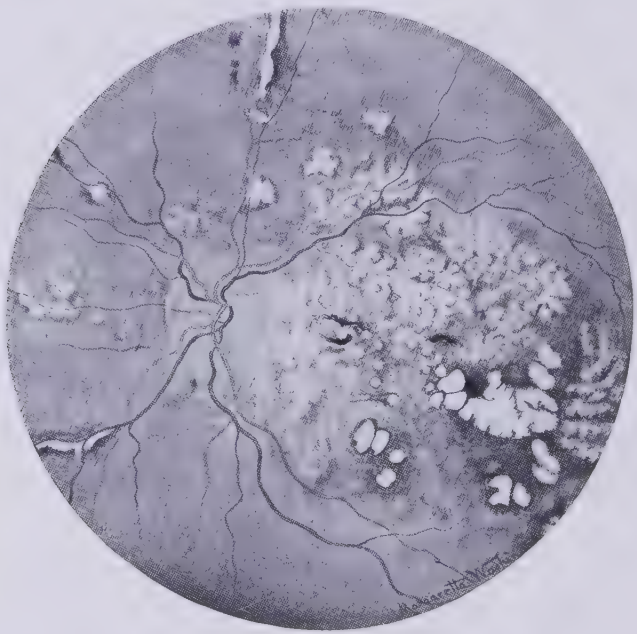


Fig. 225.—Diabetic retinitis; unusual extensive white exudations in the macular region.

lesions in the majority of the cases of diabetes. But Foster Moore is convinced there is good reason for identifying a special form of retinitis due to diabetes. The vital *prognosis* is not usually so grave as that in renal retinitis.

To a striking ophthalmoscopic picture characterized by a very light salmon color of the blood in the retinal arteries and a light chocolate hue of the veins, which are enlarged, and by some pallor of the general fundus the name *lipemia retinalis* has been given (Heyl) or *lipemia angioretinalis* (M. Cohen). These appearances are due to the presence of fat in abnormal amounts in the blood. The fat in the blood must exceed 4 per cent. before lipemia appears, and rise to about 8 per cent. before the typical picture is evident (Wagener). No hemorrhages

or exudations develop; in this respect it differs from leukemic retinitis. It is apt to occur in young diabetics. It may be transitory and may disappear when the blood-fat lessens. It has been reported in other conditions—phosphorus-poisoning, alcoholism, and nephritis.

Treatment.—There is no local treatment of diabetic retinitis. The discovery of such a condition may lead to the finding of sugar in the urine, but more commonly the patient is already conscious of his disease and is under medicinal and dietetic treatment. Recent reports indicate that *insulin* in suitable cases may be of service.

Leukemic Retinitis.—The retinal changes seen in splenic leukemia, to which variety of the disease they are almost exclusively confined, affect both eyes, usually one more than its fellow.

The most important ophthalmoscopic appearances are slight swelling of the papilla, pallor of its surface, veiling of its edges, and some opacity of the retina, especially along the lines of the vessels. The latter present a striking appearance. The veins are broad, distended, and of a somewhat rose-red color; the arteries, in contrast, narrow and orange yellow, which color substitutes the ordinary fiery red of the choroid, the vessels of which present a yellowish-red tint.

Very prominent lesions are white spots with red borders, especially near the equator and in the region of the macula lutea. The spots vary in size and are often somewhat elevated. They are due to a collection of lymph-corpuscles, and the red border to an extravasation of blood-corpuscles.

On the other hand, retinitis associated with leukemia may not present characteristic appearances, but may consist simply of a diffuse opacity of the retina, or appear in the form of hemorrhagic retinitis. When the yellow spots which have been described develop in the macula they resemble the lesions produced by albuminuria. Indeed, albumin in the urine may be present. In any doubtful case a careful blood examination will reveal the true nature of the disease.

Proliferating Retinitis.—This affection is characterized by dense masses of bluish-white or white color, which are developed from the retina and stretch out into the vitreous humor. They often cover a considerable portion of the fundus and hide the papilla, which may with difficulty be seen through the intervening spaces. Sometimes the masses follow the course of the blood-vessels, which in part may lie beneath them, and in part pass over them; those which lie above the masses are occasionally newly formed blood-vessels. As complications there may be detachment of the retina, and opacity of, and hemorrhage into, the vitreous. Vision is usually, but not always, greatly impaired; sometimes totally lost.

According to Weeks, an essential of this disease is the production of membranes which extend from the retina into the vitreous humor, and a fibrinous exudation or hemorrhage must first occur before these membranes can be formed. This process and that of vascular veils in the vitreous (see page 476) are similar. Thus a blood-clot may organize, forming a sheet or mass of fibrous tissue which is vascularized

by newly formed blood-vessels derived from the retinal system. This tissue by preference is situated near the disk because there is more mesoblastic tissue at this position than elsewhere in the fundus (Parsons).

Causes.—The relation of recurrent retinal hemorrhages (hemorrhages into the vitreous) to proliferating retinitis has been described (page 474) and its development in diabetes and nephritis has been referred to. Syphilis is undoubtedly a cause in certain cases and the evolution of this affection as part of tuberculosis of the retina is described on page 503. To anemia, chlorosis, arteriosclerosis, menstrual disturbances and the hemorrhages which they cause it has also been ascribed.

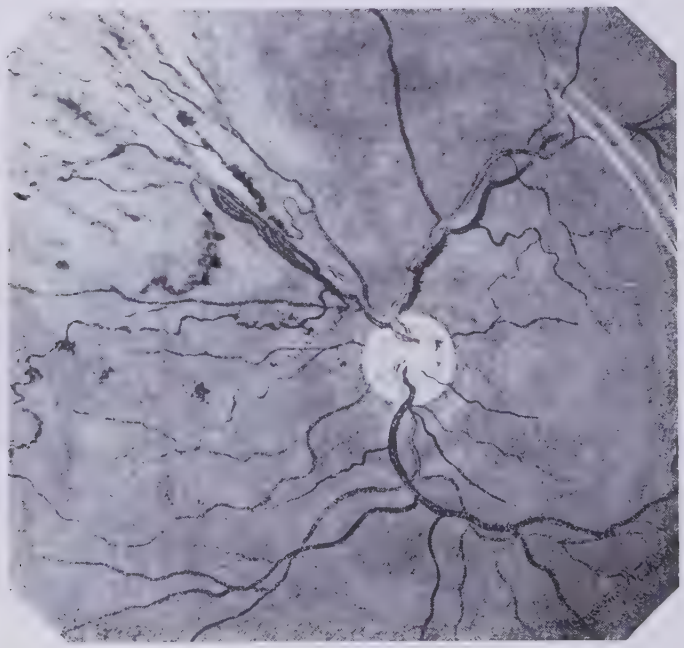


FIG. 226.—Proliferating retinitis in an early stage due to tuberculosis. Note the beginning formation of membranes extending from retina into vitreous.

A number of the cases are due to *traumatism*—penetrating wounds of the globe, concussion of the eyeball causing extensive hemorrhage, and retained intra-ocular foreign bodies.

During warfare, as illustrated in the past war, various *primary* intra-ocular lesions are produced by concussion, contusion, or impact of missiles. They result in *secondary* lesions, the most important being *atrophic chorioretinitis* (spots of atrophy, exposed scleral areas and pigment distribution, heaping and fringing), and *proliferating chorioretinitis*. If the extravasations in the retina and choroid are absorbed, many of the well-known appearances of pigmented atrophic chorioretinitis develop; frequently its elaboration is most extensive,

especially in fan-shaped, pigmented granular areas. Blood may escape, and often does, into the vitreous and may be absorbed, leaving all manner of opacities in its place. Proliferating chorioretinitis may follow, and this chorioretinitis is essentially a cicatricial process; there is organization of hemorrhage, but this is of less importance than its irritating effect on the connective tissue of the retinochoroidal layers, inciting active proliferation and the formation of tracts, areas, and masses of fibrous tissue. The whole process and picture may differ materially from the so-called proliferating retinitis of recurring hemorrhages in the vitreous and retina, especially in young subjects, often noted in civilian practice. In the type which follows war injuries the retina and choroid have been ruptured, and the cicatricial process results in a pinning down of the retina by opaque, plastic-looking material rather than in its detachment, so frequent in the ordinary variety, in which the proliferation arises from extravasated blood, and the numerous membranes, following the vessels, often partly translucent, protrude freely into the vitreous.

Treatment.—This has in part been described (page 475). Should syphilis be shown to be an etiologic factor, the usual remedies are indicated. In tuberculous varieties of the affection treatment with tuberculin may be tried and has in some instances achieved success; it must be used with caution, as it has been followed in a good many cases by severe reaction and increase of hemorrhages due to violent local reaction. Iodids and other alteratives, diaphoresis and diuresis are worthy of trial; fibrolysin has been referred to.

The prognosis is most unfavorable if the disease is *extensive;* fresh hemorrhage and fresh proliferations are only too common. The lesions of traumatic proliferating chorioretinitis are practically unaffected by treatment. Detachment of the retina and sometimes shrinking of the eyeball occur.

Tuberculosis of the Retina.—The relation of tuberculosis to various retinal affections, namely, recurrent retinal hemorrhages, retinal periphlebitis, and proliferating retinitis (pages 474 and 501), has been discussed.

In some recent studies of this subject Edward Jackson and W. C. Finnoff conclude that retinal tuberculosis begins by the formation of white infiltrations in front of retinal vessels, generally the veins. At a later period perivasculitis and hemorrhages are manifest; the hemorrhages may disappear and may be completely absorbed if they are small and confined to the retina, or they may be large, massive, and burst into the vitreous. In these circumstances the well-known appearances of proliferating retinitis (Fig. 226) develop. White spots, something like those seen in renal retinitis, may arise and add to the depreciation of vision. In a case studied recently by the author the typical white infiltrations, three in number, appeared along the superior temporal vein, which was irregularly contracted. A sharp exacerbation followed a tuberculin injection, when numerous fresh white spots appeared in front of this vein and on some of its connecting branches

with minor areas of retinal hemorrhage; ultimately they subsided. The course of the disease may be protracted.

Treatment.—In the absence of pyrexia and extensive tuberculosis elsewhere in the body, Jackson and Finnoff recommend tuberculin, which, however, must be very cautiously given. General supporting measures are indicated.

Retinitis Circinata.—This name was given by Fuchs to an affection characterized by a concentric aggregation of slightly raised white spots and lines around the macula. Sometimes the white spots surround the macula after the manner of a wreath; sometimes the arrangement is more like that of an ellipse, one end of which may touch the edge of the optic disk, while the other extends beyond the macular region (Lawford). Fuchs regards the white patches as fibrinous exuda-

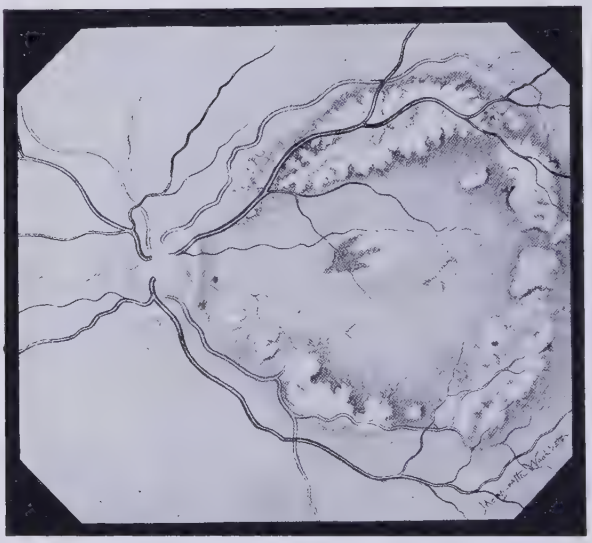


Fig. 227.—Circinate retinitis (from a patient in the Jefferson Hospital).

tions which have taken place into the deeper layers of the retina, while de Wecker denied the special character of the disease, which he attributed to fatty degeneration, the result of hemorrhages. Indeed, Amman has shown that the white spots are due to fatty cells clustered where hemorrhages have been. Hemorrhages may accompany the affection, and in one case (Fridenberg) there was a development of new-formed blood-vessels in the retina. The lesions have also been attributed to disease of the smallest macular vessels, especially the arteries (Oeller), and also to the results of a long-standing edema, the size of the circle varying according to the extent of the previous affection (Gunn). Sometimes the disease is essentially chronic and the appearance remains unchanged for years; sometimes it is slowly but surely progressive, and rarely the ring of exudate may partly or entirely

disappear. Such disappearance within one year has been noted by N. Bishop Harman.

Retinitis Striata.—Occasionally light or yellowish-white stripes extending from the periphery toward the disk, and sometimes bordered by lines of pigment lying beneath the retinal vessels, are apparent to the ophthalmoscope. To this appearance the name *retinitis striata* has been given, and while the origin of the affection is not positively known, Holden contends that the stripes are the result of the metamorphosis of retinal hemorrhages, and in this respect are analogous to angioid streaks. On the other hand, it has been maintained by L. Caspar that these retinal striations represent the final stages of spontaneously cured detachments of the retina. The author has published the case histories of two patients which appear to confirm this view of the origin of retinitis striata.

Pigmentary Degeneration of the Retina (*Retinitis Pigmentosa*).—Although this affection is usually entitled *retinitis pigmentosa*, the phenomena of inflammation are absent, and it consists of a degeneration of the retina, associated with great contraction of the blood-vessels and the accumulation and deposition of pigment from the pigment epithelium of well-nigh characteristic form in the substance of the retina. Both eyes are always affected.

Symptoms.—The ophthalmoscopic appearances of a typical case are as follows:

(*a*) *Pigmentation.*—The pigment masses frequently assume an appearance resembling bone-corpuscles, and by the union of their processes suggest the Haversian canals. The resemblance of the pigment to bone-corpuscles is not always evident; the pigment deposits may be round and irregular and simulate the pigment spots of choroiditis, but unlike them they are situated in front of the blood-vessels and are in the inner layers of the retina. By preference, the pigmentary deposits are more marked on the temporal side. They begin in the periphery of the eye-ground, although not usually in the extreme periphery, often lying along the course of the main vessels, which may be in places encrusted by them, and gradually approach the papilla, the macular region remaining for a long time unaffected. A zone midway between the center and far periphery is the favorite seat of pigmentation.

(*b*) *"Wainscoted" Fundus.*—A perfect picture of the appearance already described in connection with superficial choroiditis is visible on account of the absorption and decolorization of the retinal pigment epithelium and the exposure of the larger vessels of the choroid. The overlying retina is distinctly gray.

(*c*) *Contraction of the Vessels.*—This is present in both systems. The vessels may be as thin as threads. Often their walls exhibit patches of opacity, and they are accompanied by fine white lines and covered here and there by pigment deposits. Not only are they greatly contracted, but they are apparently diminished in number.

(*d*) *The Changed Nerve-head.*—The color of the papilla, according to the stage of the disease, is of a yellowish-gray, yellowish-red, or waxy

tint. It finally becomes dull white and atrophic. Except a slight veiling, its edges are plainly marked.

(*e*) *Opacities of the Media.*—Cataract at the posterior pole is frequently present, and in the later stages posterior cortical cataract (see Fig. 200). Opacities in the vitreous are uncommon.

(*f*) *Nystagmus.*—Quite frequently a quick lateral oscillation of the eyeballs, or nystagmus, is present, especially in congenital cases.

The subjective symptoms are:

(*a*) *Depreciation of Central Vision.*—Visual acuteness may be but slightly affected in the earlier stages, although usually the perception of green and red is below the normal. Indeed, reasonably good central vision may remain even when the disease is very widespread, but it finally sinks with the progress of the affection and, ultimately, blindness results, although this usually does not occur until the expiration of many years.

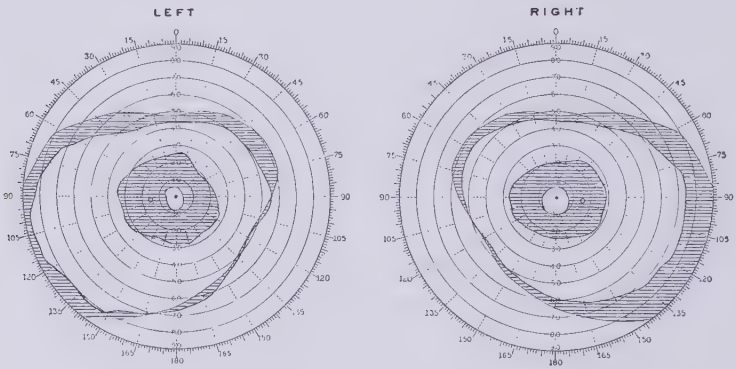


Fig. 228.—Ring scotomas in pigmentary degeneration of the retina.

(*b*) *Contraction of the Field of Vision.*—In the early stages of pigmentary degeneration of the retina the peripheral field of vision may be nearly normal in extent, if the illumination is good, although much contracted if the illumination is reduced. Later, the field contracts concentrically, according to the amount of degeneration, and the contraction may be so excessive that only a very small area of the field remains. In rare instances, even with extreme narrowing of the visual field, there is still moderately good central vision, and the patient may read by fixing a single word at a time. Finally, the contraction goes on to complete blindness. As the *extreme* periphery of the retina is often free from pigmentation in the earliest stages of the disease, when the equatorial region is already involved, the periphery of the visual field may be intact, but between it and the preserved central field there is a blind zone; that is, an *annular* or *ring scotoma.* Indeed, the earliest manifestation of pigmentary degeneration of the retina is this ring scotoma (M. L. Hepburn). At first it is incomplete and represents the loss of function in the intermediary zone of the

retina. Later other portions of the retina degenerate in regular order, the fixation point being the last to disappear.

(*c*) *Night-blindness.*—Usually this is the first symptom which calls attention to the case. The patient is uncertain in his movements and stumbles as soon as twilight begins, becoming quite helpless in the dark. Pronounced night-blindness is not always present, and in rare instances diminished light is a relief to the patient. Such a condition is due to retinal hyperesthesia.

Atypical Varieties.—The pigment may be massed in the macular region; the central vision is much affected, and a scotoma appears around the point of fixation. In other instances the pigment is scattered all over the fundus in irregular masses, and is associated with clear, shining spots lying beneath the retinal vessels.

Cases occur presenting the usual subjective symptoms, but without the accumulation of pigment—really forms of *sclerosis of the retina without the formation of pigment*, and constitute the so-called *pigment degeneration without pigment*. Even in the very beginning of pigmentary degeneration of the retina there may be no pigmentation visible to the ophthalmoscope. But in the cases referred to migration of pigment into the retinal substances is not observable for many years, indeed, it may not appear at all, although waxy disk degeneration and contracted vessels are evident. Therefore pigmentation is not necessarily an essential condition of the disease. A diffuse opacity of the retina has been observed in some of these cases (Leber).

Retinitis Punctata Albescens.—This type of retinal affection was originally described by Mooren, and, according to him, is characterized by a great number of striæ or spots scattered over the fundus, resembling in color the reflex of the sclera. The retinal vessels are not covered by the spots. The papilla is decidedly gray. A relative or a positive scotoma may be present. To a similar retinal disease the name *central* and *punctate retinitis* has been given (Hirschberg). Fuchs has called attention to the similarity of this disease to retinitis pigmentosa, inasmuch as it is either congenital or starts in infancy, affects several members of the same family, and may occur in the children of blood relations. Also, there may be night-blindness and contraction of the visual field. It should be regarded as a primary degeneration of the retina and choroid allied to pigmentary degeneration of the retina, and should not be classified as an inflammatory disease. Leber classified the condition as a form of non-pigmented tapetoretinal degeneration and believed that the white spots represented partly calcified colloid excrescences of the lamina vitria (druses). Nettleship, however, was not willing to admit this origin of the lesions.

Another type of chronic retinal degeneration is that to which Fuchs gives the name *atrophia gyrata choroideæ et retinæ*, also seen in the children of consanguineous marriages and associated with night-blindness. There is extensive atrophy of pigment epithelium of the choroid, which may be peripherally situated and appear as a white zone with an irregular border, or nearly the whole fundus, except an area around the

macula, may be involved (*choroideremia*, see page 384). Generally the pigmentation present is identical with that of retinitis pigmentosa.

Cases of pigmentary degeneration of the retina with lesions especially located in the macular region are, according to Nettleship, not very uncommon, the morbid process being either congenital or having begun early in life. The area at the posterior pole has a granular appearance and the pigmentation is in the form of dots and not of the "bone corpuscle type." The patch of macular change may have an irregular but defined outline, or blend with the surrounding retina which apparently is not diseased; or beyond there may be a zone of typical pigmented retina. For lesions of this and similar character, attributed by him to abiotrophy of the retinal neuro-epithelium, Collins suggests the designation *pigmentary macular retinal degeneration*, or *retinitis pigmentosa of the macula*. (For other types of macular retinal degeneration, see pages 537, 538.)

Complications.—Retinitis pigmentosa may be complicated with *chronic glaucoma*, the retinal affection probably antedating the glaucoma. In one family several members of which have typical pigmentary degeneration of the retina the author has observed two with glaucoma, in one instance assuming a subacute type successfully controlled by operation. Holloway has described pigmentary degeneration of the retina in association with renal neuroretinitis. In addition to cataract, nystagmus, strabismus, deafness (33 per cent. [Nettleship]), harelip, supernumerary toes and fingers, and other congenital anomalies occur. *Polydactylism* and *pigmentary degeneration* of the retina may be associated with disorders of the pituitary body. In addition to the retinal disease and supernumerary fingers and toes, atresia ani and malformations of the skull may be present; also retarded mental development, marked obesity, with or without genital hypoplasia, and disturbances of digestion.

Causes.—The disease is markedly hereditary. Simeon Snell has reported the history of this affection in five generations, 28 of the 67 descendants being affected. Consanguinity of the parents of the subjects of this disease has been found in a certain number of cases; indeed, the disease has been attributed to this cause alone. Nettleship's results are as follows: Heredity without consanguinity in 23.5 per cent., consanguinity without heredity in 23 per cent., and heredity combined with consanguinity in 3 to 4 per cent. Hereditary syphilis has been suggested as a possible cause of retinitis pigmentosa, but this etiology has not been proved. The affection is found among deaf mutes (4 per cent. [Nettleship]), idiots, and epileptics, and in this sense is connected with morbid states of the nervous system. Very often no cause can be assigned. The disease is either congenital or usually begins in childhood; it may, however, arise at any age of life. According to W. T. Shoemaker, it is always congenital in origin, no matter how late its manifestations are evident. According to Nettleship acute exanthemata, tuberculosis, excessive loss of blood and syphilis may determine the onset of the disease if in the choroid and retina a predisposition exists.

Pathology and Pathologic Anatomy.—A degenerative process begins early in the outer layers of the retina, which adhere to the choroid. The rods and cones disappear, the blood-vessels, choroidal and retinal, are sclerosed, those of the choriocapillaris being the first affected, and their lumens contracted. There is a marked infiltration of pigment cells, the pigmentation being a secondary change produced because the blood-supply is checked. Later the degenerative process reaches the layer of ganglion cells and the nerve-fibers. The optic nerve atrophies and may contain hyaline masses similar to those in the lamina vitrea of the choroid. Wagenmann believes that the primary lesion is a sclerosis of the vessels of the choroid. Gonin and Nettleship



Fig. 229.—Pigmentary degeneration of the retina; sclerosis of choroidal vessels (from the William F. Norris collection in University of Pennsylvania).

attribute the primary degeneration to deficient blood-supply owing to obstruction to the blood-current in the choriocapillaris. The beginning of the disease has also been placed in the pigment epithelium. The affection is always bilateral. The statements thus far recorded are in brief summary the ones usually made with reference to the pathology and pathologic anatomy of retinitis pigmentosa. Quite recently E. Treacher Collins has advocated an entirely new theory. The chief argument, quoting Collins, against vascular sclerosis being the primary cause of pigmentary degeneration of the retina is that typical cases have been investigated in which no changes in the choroidal blood vessels were found in the affected eyes microscopically examined. He suggests that the primary change is *abiotrophy*, that is, degenera-

tion of tissues due to defective vitality of the retinal neuro-epithelium, which, he believes, explains all the symptoms of the disease.[1]

Diagnosis.—Retinitis pigmentosa may be distinguished from disseminated choroiditis by the difference in the pigmentation of the two diseases.

Its differential diagnosis from certain types of pigmented retinochoroiditis in acquired syphilis is difficult, especially where the latter manifest themselves in a form of atrophy of the retina and a gathering of pigment spots, beneath which the exposed choroidal vessels are visible. In retinochoroiditis, however, the pigment spots do not have the characteristic form: they are much scattered, and do not follow or cover the blood-vessels; besides, vitreous opacities, which are comparatively rare in pigmentary degeneration of the retina, are usually present. The visual field should lend aid in diagnosis, as in choroidal disease the characteristic feature is the patchy nature of the scotomas (M. L. Hepburn).

A patient with night-blindness, or seen stumbling about during the twilight, should be subjected to a careful examination of the periphery of the eye-ground after dilatation of the pupil, because occasionally the pigment is confined to this region and might otherwise be overlooked. Sclerosis of the retina without pigmentation must be kept in mind and the clinical and family history carefully studied, as well as the visual field.

Course and Prognosis.—Pigmentary degeneration of the retina almost always progresses steadily onward with ever-increasing contraction of the field of vision, until finally, usually by middle life, sight has been obliterated, with, perhaps, the exception of a slight eccentric preservation of the field. According to Nettleship, the age at which blindness becomes complete is variable, and, with rare exceptions occurs only after the thirtieth to the thirty-fifth year of life; more usually after sixty years. The prognosis is, hence, nearly always unfavorable in all circumstances and in spite of all known endeavors to modify the course of the disease. Occasionally congenital cases remain stationary. Sometimes when the pigment accumulation has advanced far over the retina, but the macula is still free, the disease remains stationary for long periods of time, and good vision within the narrow field continues.

Treatment.—Strychnin in full doses, especially by the hypodermic method, is frequently employed, and recently thyroid extract has been advocated (Jones). If there is any suspicion of syphilitic taint, the usual remedies are indicated. Galvanism has been tried, and under its influence, it is stated, the progressive contraction of the field of vision has been stayed, although no improvement in the acuteness of central sight was obtained. It certainly should be given a trial in every case. It is always important, considering the slow advance of many of the cases of retinitis pigmentosa, to correct refractive error carefully; there

[1] Transactions of the Ophthalmological Society of the U. K., 1919, vol. xxxix, p. 165.

may be present considerable degrees of astigmatism and suitable glasses not infrequently are of advantage in improving central vision. Iridectomy has been tried and paracentesis of the anterior chamber. An encouraging result in increasing the size of the visual field by corneoscleral trephining has been obtained; in one case night-blindness disappeared and remained absent for some months (Mayou). Although the posterior cortical cataract of this disease rarely reaches maturity, the lens should be extracted, as vision may be thus materially improved (Doyne, Knapp). The author has had a few excellent results by adopting this procedure.

Sometimes a peculiar *pigmentation of the retina* is encountered which has been mistaken for a type of retinitis pigmentosa, with which disease, however, it has no alliance. The pigment changes occupy a sector of the eye-ground, and consist of collections of black or chocolate-brown spots, with a strong tendency to group formation. The spots may be round, oval, and often suggest diminutive bunches of grapes. This appearance was figured by Jaeger, has been described by Juler, Stephenson, and others, and, quite recently, elaborately by Hoeg. It is doubtless a congenital condition; the function of the eye is undisturbed. The author has studied a number of cases of this character and no increase in the lesions has been noted, although several of the patients have been under observation for years.

Detachment of the Retina (*Ablatio Retinæ; Amotio Retinæ*).—Separation of the retina from the underlying choroid (so-called spontaneous detachment) is due to an accumulation of a serous fluid between these membranes (*serous detachment of the retina*).[1]

Symptoms.—The student will observe, as he examines the various portions of the fundus with the ophthalmoscope (direct method), an alteration of refraction at the area of separation, the surface of the elevation thus produced being out of focus as compared with the rest of the eye-ground. Thus, if the general fundus is hyperopic, the detached portion is more hyperopic, and requires a stronger convex glass for the study of its surface; if it is highly myopic, a weaker concave glass, or, it may be, a convex lens.

The normal color of the fundus is lost as the detached retina is approached, which appears as a gray or bluish-gray membrane stretching forward into the vitreous, containing folds which give rise to a sheen. The intervening furrows present a greenish-gray reflex, and the whole oscillates with the movements of the eye if the underlying substance is fluid; if it is a solid, neither folds nor tremulousness are present. Rents and holes in the detached retina, through which the choroid is visible, are often demonstrable. These holes may be due to trauma, to cystic degeneration of the retina, and to choroiditis.

The retinal vessels rising over the separated portion first lose the

[1] Sir William Lister points out that the term "detachment of the retina" is a misnomer, in that the condition usually so described is actually a separation between the retinal pigment epithelium and the percipient neural portion of the retina. (Consult Amer. Jour. Ophth., 1923, 6, 1009.)

light-streak, and finally appear as dark, tortuous cords. They apparently are of smaller size than normal, and if followed backward they pass out of focus at the sloping edge of the detachment; indeed, there may be a yellowish border and occasionally accumulated pigment. The amount of discoloration of the detached area depends upon whether the case is recent or not, and upon the character of the underlying substance. In the earlier stages transparency is not lost, and the gray color, previously described, may not be present.

The detachment, either *partial* or *complete*, may occupy any portion of the fundus, but commonly is found below, preceded, it may be, by a separation of the retina in the upper part of the eye-ground. These superior detachments may sag downward and bulge forward into the vitreous like a diminutive balloon; they may overhang and partially obscure the optic disk. Not infrequently a superior detachment is transferred to an inferior position. The subretinal fluid seeps to the lower area and the originally separated portion of the retina is reattached. Sometimes the detachments are quite small, like a series of furrows, and at other times an almost circular circumscribed separation occurs. Finally, the subjective signs of detachment may be present without discoverable elevation of the retina, but over the area (which subsequently separates) there is complete loss of the light reflex from the retinal vessels (Loring). In these circumstances the visual field should be mapped under reduced illumination. At first, owing to the small quantity of subretinal fluid, the detachment may be *flat;* occasionally it remains so, but usually it increases in extent and elevation.

Unless the macular region is directly involved, vision is not obliterated, but there is always interference with sight. This may develop suddenly. The field of vision is lost in an area corresponding to the detached retina, and the completely darkened portion is usually bordered by a zone of imperfect vision corresponding to an area of retina not yet separated, but elevated above its normal plane. If the retina is detached below, the upper portion of the visual field is obliterated; if above, the lower portion, and so on (Fig. 231). A retinal detachment just beginning may not be detected by a visual-field examination with a white test-object, but may be represented by a relative scotoma if the test-object is blue.

The patients are conscious of distortion of objects (metamorphopsia); of floating spots before the eyes, due to the frequent presence of vitreous opacities; of an appearance like a cloud, due to the scotoma produced by the separated area; and of phosphenes, although the last cannot be elicited by pressure on the eyeball over the separated area and sometimes of monocular diplopia. Should a patient with high myopia describe "attacks" of flashes of light, showers of sparks, or other types of photopsies, such symptoms are dangerously significant of impending retinal detachment. Exteriorly the eye ordinarily presents no abnormalities. Sometimes the anterior chamber is deep; the tension may be diminished. In late stages of retinal detachment the vitreous

becomes exceedingly cloudy owing to increasing the vitreous opacities; cataract frequently forms and becomes complete. Iritis and iridocyclitis may develop, and although the tension is usually lower than normal in detachment of the retina, if seclusion of the pupil takes place as the result of iridocyclitis it may be elevated.

Causes.—The causes of ordinary retinal separation are: Myopia (61 per cent. in Uhthoff's material) either of moderate or high degree, more frequently the former than the latter, according to Lister; traumatisms

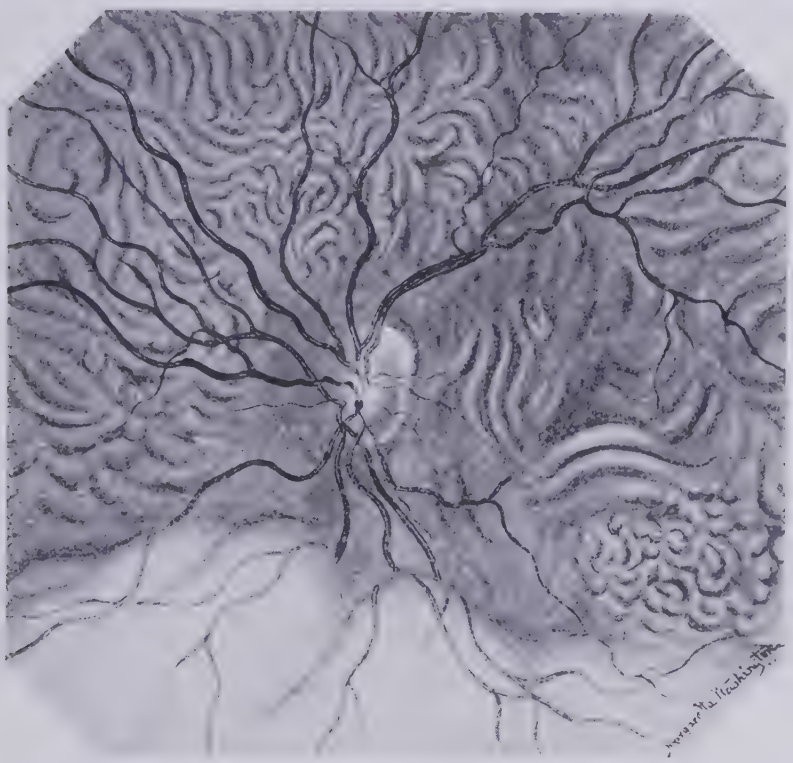


FIG. 230.—Traumatic detachment of retina below; above the retina is less completely detached and is thrown into numerous folds (from a patient in the University Hospital).

and effusions of blood, preceded usually by hemorrhages into the vitreous or retina. More men than women acquire simple detachment of the retina; myopic refraction most frequently is present, and the separation is more apt to occur in an eye in which the visual disturbance has rapidly developed. The condition often becomes apparent suddenly, especially after physical exertion; occasionally it develops gradually and the obscuration of vision has been compared to a curtain drawn over the sight. Antedating the actual separation, the patient may be conscious of flashes of light and muscæ volitantes. Sudden detachment cannot occur without a hole or rent in the retina (Lister).

Retinal detachment is also caused by intra-ocular tumors (sarcoma of the choroid, metastatic carcinoma, glioma), subretinal parasites (cysticercus), tumors and abscesses in the orbit, and diseased conditions of the eye, as retinitis, especially renal retinitis, retinitis proliferans, and the retinitis of pregnancy, cyclitis, iridocyclitis, etc. In iridocyclitis the detachment is often found only after removal of the shrunken globe, and is caused by contraction during organization of strands of connective tissue attached to the retina. Separation of the retina may be *congenital*, due to syphilis, and it has been observed in several members of the same family (*hereditary detachment of retina*—Pagenstecher). Subretinal hemorrhage—the blood coming either from the retina or choroid—may produce the so-called *hemorrhagic detachment of the retina*, for example, in the subjects for arteriosclerosis, but also in anemia and chlorosis. In *traumatic detachment* of the retina the eye is often, but not necessarily, myopic; the separation may immediately follow the injury or be delayed for weeks or even months. The frequency with which a history of injury long antedating the actual retinal separation may be obtained is emphasized by Harold Gifford. The cicatrix which follows a penetrating wound of the sclera, fastening as it does the retina to the choroid and sclera by later contraction, may detach the retina.

Mechanism.—Leber and Nordenson taught that the first process is a fibrillar change in the vitreous, which shrinks and occasions traction (retraction theory). This ruptures the retina, and the fluid from the vitreous cavity passes beneath it through the opening. The primary cause of the pathologic alteration in the vitreous is believed to be disease of the choroid and ciliary body. Greeff, Elschnig, and others, however, doubt if retinal detachment is caused by shrinking of the vitreous; detachment of the vitreous they regard as an artefact due to the action of fluids used in hardening the eyeball (see page 478). Raehlmann explained the detachment by a *diffusion theory*, the conditions being analogous to transudations in other parts of the body. In some instances retinal detachment must be explained by the presence of exudation or hemorrhagic extravasation, and in axial myopia of high degree the "mechanical theory" is applicable—the separation of the retina being due to elongation of the eyeball, fluidity of the vitreous, and disease of the choroid.

Lister summarizes the mechanism of detachment of the retina as follows: The retina may be pushed in by fluid secreted from the choroid into the interretinal space, as in neoplasm, choroiditis, and retinitis; or pulled in by adherent and contracting vitreous, vitreous bands, or cicatricial changes in the retina (*retinitis striata*); or floated in by transposition of fluid vitreous through a hole in the retina.

Diagnosis.—No difficulty arises in detecting a large detachment of the retina by attending to the symptoms already detailed. An extensive or complete detachment which floats far forward may be examined by oblique illumination. If the vitreous is full of opacities, a study of the field of vision is useful. If the substance underlying the

detached portion is *fluid*, there are usually diminished tension of the eyeball and the appearance of furrows in the separated tissue, which trembles with the movements of the eye, symptoms which are absent if a *solid growth* has caused the separation; also a growth can be excluded if a hole in the retina is visible and if the loss of sight has been sudden (Lister; see also page 405). Important diagnostic signs are the loss of the light-reflex of the vessels, and their dark color over the area of separation. They can be seen to regain the light-reflex in passing over the normal retina. The diagnosis of *flat detachment of the retina* is often difficult. It is important to submit all eyes with detachment of the retina to the transillumination test (see page 405).

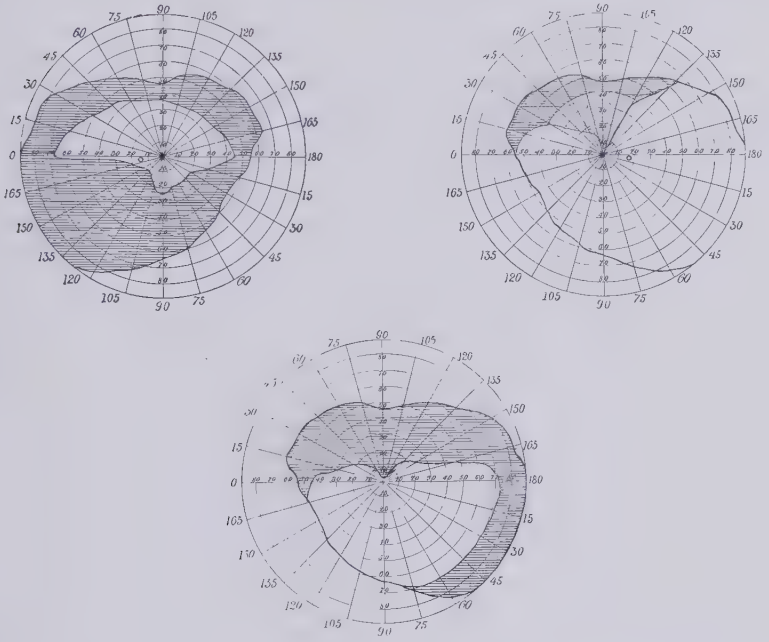


FIG. 231.—Various types of fields of vision in detachment of the retina.

Prognosis.—This is not favorable, and many of the suggested means of treatment have proved unsatisfactory. Even though they should be followed by reattachment and the function of the retina should be restored, the good effect is only too frequently not a lasting one because vitreous disease continues. However, good and permanent results have been secured and suitable measures should be given full trial. Duane, quoting Leber, says a cure in the sense of a reattachment takes place in about 8.5 per cent. of the cases, but the restoration of even moderately useful vision only in from 3 to 6 per cent. of the cases. In a few instances *spontaneous reattachment of the retina* takes place. In the reattached area spots of choroiditis are often visible and variously disposed striæ, white or fringed with pigment (see page 531).

Treatment.—This should include rest in the prone position, a pressure bandage, preferably elastic, and pilocarpin sweats, the pupil of the affected eye being dilated with atropin, and lymphagogue activity stimulated by the use of a 5 per cent. dionin solution. Internally, the various iodids or small, frequently repeated doses of salicylate of sodium may be administered.

Various forms of operative procedure have been attempted: sclerotomy and, comparatively recently, trephining the sclera, thus evacuating the subretinal fluid; evacuation of the subretinal fluid by puncture and aspiration, and drainage by means of a gold wire; electrolytic puncture; incision of the fibrous bands in the vitreous, followed by the injection of the vitreous humor of a rabbit (Deutschmann); resection of the sclera (Müller). Subconjunctival injections are valuable. For this purpose de Wecker employed a solution composed of $3\frac{1}{2}$ parts of gelatin with 100 parts of physiologic salt solution. Jocqs advocates injections of a saturated solution of salt in conjunction with scleral puncture, while Bourgeois recommends a 30 per cent. salt solution to which a few drops of a 5 per cent. solution of cocain are added, 1 c.c. of the fluid being injected. Subconjunctival injections of cyanid of mercury and of sodium chlorid are also advocated; they may be rendered painless by adding acoin to the solution. The author has secured a few favorable results with scleral puncture, followed by large (30 minims—1.9 c.c.) injections of physiologic salt solution, and has not found it necessary to increase the strength of the salt solution beyond 4 or 5 per cent. During the treatment the patient should remain in bed. In place of subconjunctival injections of salt solution, sodium citrate (4 per cent., diluted with 2 parts of physiologic salt solution) may be used. Scleral cauterization, followed by subconjunctival saline injections, has been employed by Dor, and puncture of the eyeball with the galvanocautery has been advocated. With Deutschmann's operation (bisection of the vitreous and retina, with or without the intravitreous injection of animal vitreous) the author has had no experience. Deutschmann reports good results; some surgeons who have investigated the subject are not in favor of the operation. If the detachment is due to a tumor, the eye should be enucleated (see page 406).

Hemorrhages in the Retina (*Apoplexy of the Retina*).—The appearances of retinal hemorrhage have been described in the general symptom-grouping, and as they occur with so-called hemorrhagic retinitis (see page 492).

Hemorrhages (unassociated with inflammation) may take place in any of the layers of the retina, or, bursting through the limiting membrane, they may occupy the vitreous humor. By preference they are found along the course of the larger vessels; a favorite site is the macula.

Hemorrhages originating in the outer sheath of the optic nerve may appear at its margin and spread into the surrounding retina, although it does not follow that such hemorrhagic extravasations always follow this course.

Hemorrhages of large dimensions and drop-like form usually mean an extravasation between the internal limiting membrane of the retina and the hyaloid membrane of the vitreous, and they come from a retinal vessel. These *subhyaloid* or *preretinal hemorrhages* tend to occur at the yellow spot more than at other parts of the fundus. Occasionally they assume a wedge-, bottle-shaped, or almost circular appearance, or they may have, as in an eye recently examined by the author, a long, roll-like form, and overlie the sweep of the retinal vessel; often they are semilunar in shape. Except as the result of traumatism they are exceedingly scarce in young children, but Harms has reported one case in a child of four and one-half years. According to J. Herbert Fisher, the hemorrhage detaches the internal limiting membrane from the retinal layers, which are not invaded, and occupies the space thus formed. It may break into the vitreous.



Fig. 232.—Retinal hemorrhages (de Wecker and Masselon).

Causes.—Some of these have already been enumerated. The following résumé, based upon the classification of Dimmer, which has been somewhat elaborated, may be added:

(*a*) Hemorrhages caused by changes in the composition of the blood and the tissues of the blood-vessel walls: Pyemia, septicemia, ulcerating endocarditis; diseases of the liver, spleen, kidney, and atheroma of the vessels, and angiosclerosis of the retina; loss of blood (menorrhagia, hematemesis); anemia (simple and pernicious); hemophilia, purpura, and scurvy; diabetes and gout; tuberculosis; malaria, and recurrent fever. In carcinoma of the stomach retinal hemorrhages and white spots may resemble those seen in pernicious anemia. To this manifestation the name *cachectic retinitis* is sometimes applied. Focal infections may be responsible for retinal hemorrhages.

(*b*) Hemorrhages caused by disturbances in the circulation: Hypertrophy of the heart and stenosis of the valves; thrombosis of the central vein of the retina, and embolism or thrombosis of the central artery; suffocation, compression of the carotid; retinal hemorrhages in newborn infants are not uncommon.

(*c*) Hemorrhages caused by sudden alterations of the intra-ocular tension—*e. g.*, after iridectomy or corneoscleral trephining in glaucoma—and by traumatisms. Among the latter may be classed retinal hemorrhages after large cutaneous burns, and those which have followed compression of the thorax and neck and fracture of the skull.

(*d*) Hemorrhages caused by certain toxic agents—*e. g.*, phosphorus, chlorate of potassium, serpent virus.

Prognosis.—This depends upon the extent and situation of the hemorrhages. They form an important prognostic guide of the disease

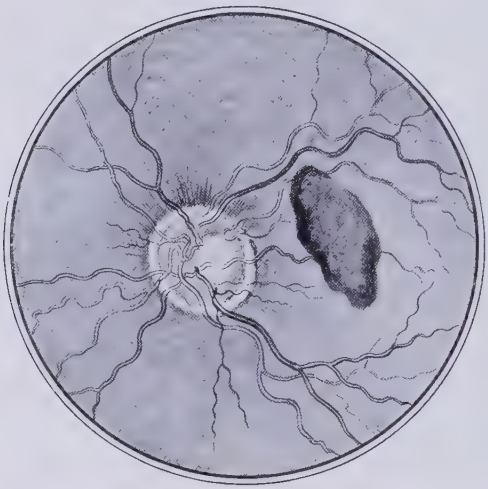


Fig. 233.—Subhyaloid hemorrhage.

which has caused them, and in elderly persons may be an indication of future hemorrhages into the brain. Glaucoma, detachment of the retina, and the formation of dense opacities in the vitreous humor may be complications.

Treatment.—All use of the eyes must be forbidden. Locally, a weak solution of sulphate of eserin may be employed, especially in elderly people to check any tendency to increased intra-ocular pressure. Internally, the medication must be governed by the probable cause. Frequently cardiac sedatives, moderate diaphoresis, and later, alteratives, such as iodid of potassium, iodid of sodium, syrup of hydriodic acid, and bichlorid of mercury, are required. If the arterial tension is high, nitroglycerin should be administered. Lactate of calcium may be tried, but should not be used continuously for more than several days at a time.

Changes in the Retinal Vessels and Their Walls.—Changes in the retinal vessels due to *vasculitis* and *perivasculitis* are of frequent occurrence. These are characterized by the appearance of white stripes along the vessels or, rather, the vessel walls become apparent by their conversion into whitish tissue, due probably to an infiltration of the adventitia composed of lymph-corpuscles. This may be so extensive that the entire set of vessels is converted into a series of branching white lines.

Such extensive conditions may be due to various diseases of the retina and optic nerve in association with general maladies, or result from certain toxemias and infections.

Alterations in the retinal vessels are also caused by *chronic nephritis*, *hypertension* (*hyperpiesis*), and *general arteriosclerosis*, and may present, according to the various conditions which obtain, the following ophthalmoscopic appearances:

1. Alterations in the course and caliber of the retinal arteries, manifesting themselves as (*a*) undue tortuosity, which is not significant unless, to quote the words of Mr. Gunn, whose classification, somewhat modified, is followed, it is associated with other evidence of disease; (*b*) alterations in the size and breadth of the retinal arteries, presenting, as it were, a beaded appearance.

2. Alterations in the reflections from, and the translucency of, the walls of the retinal arteries, manifesting themselves (*a*) in increased distinctness of the central light-streak on the retinal vessel and an unusually light color of the entire breadth of the artery ("silver-wire arteries"); (*b*) loss of translucency, so that it is impossible to see, as it is in the normal state, through the artery an underlying vein at the point of crossing; (*c*) positive changes in the arterial walls, consisting of whitish stripes, indicating degeneration of the walls or infiltration of the perivascular lymph-sheaths (*perivasculitis*).

3. Alterations in the course and caliber of the veins, together with signs of mechanical pressure, manifesting themselves (*a*) in undue tortuosity, which, as in the case of the arteries, is not significant except in the presence of other disease; (*b*) alternate contractions and dilatations ("beading" of the vessel); (*c*) an impeded venous circulation where a diseased artery crosses it. The last is a sign of the utmost importance. Ordinarily, as an artery crosses the vein, as may be seen by an examination of the normal eye-ground, there is no sign of pressure. If the walls of the artery are thickened by disease, it presses upon the vein, pushes it aside, or directly contracts its caliber, so that beyond the point of crossing there is an ampulliform dilatation. (*d*) Changes in the venous walls, precisely as they occur in the arteries, so that whitish stripes border the vessel, and are indications of degeneration in its walls. Often associated with this one may see varicosities. Proliferation of new-formed vessels may be seen. (See Plate V and Fig. 234.)

4. Edema of the retina, manifesting itself (*a*) as a grayish opacity, which may be present in the immediate neighborhood of the papilla,

or in spots over the eye-ground and along the course of the vessels, looking like a fine gray haze, or in little fluffy islands far out in the periphery and (*b*) brick red congestion of the disk, or its margins are blurred.

5. Hemorrhages, manifesting themselves as linear extravasations along the course of the vessels, roundish infiltrations scattered over the fundus, or sometimes in a drop-like form.

All these changes have been described by Raehlmann, H. Friedenwald, Hertel, Hirschberg, Foster Moore, Bardsley, Adams, Benedict, Wagener, the author, and many other observers, and were especially accurately recorded by the late Mr. Marcus Gunn. The preceding para-

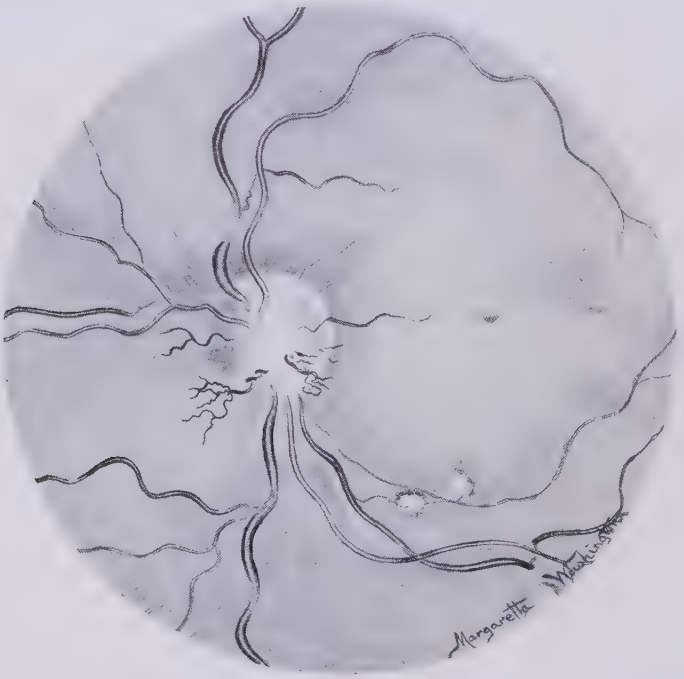


Fig. 234.—Fundus in arteriosclerosis. Perivasculitis, indented veins, newly formed capillaries on disk, hemorrhage.

graphs portray in general terms the various vascular alterations which may be seen in hypertension, angiosclerosis, etc., without especially attempting to group those which more or less exactly interpret special conditions.

From the retinal vessel changes which have been described, due to the local retinal vascular disease and distinct from renal retinitis, *angiosclerotic retinitis* develops, which has been described and classified by Foster Moore. According to him sharp-edged spots, somewhat circular in shape, unassociated with a surrounding edema, appear, sometimes in the macular region (occasionally forming a star figure) and also elsewhere. They may disappear, but slowly; fresh ones occur. Hemor-

PLATE V.



Changes in the fundus in arteriosclerosis.

rhages are evident; the arteries are bright, and their reflexes may be broken or dotted; arteriovenous compression is marked. The condition may be unilateral (45 per cent. in Moore's experience) and exist for months and years. The exudations are due to hyaline material in the outer molecular layer of the retina.

The *significance* of these lesions is of serious import. In addition to their relation to nephritis, they may be the forerunners of vascular sclerosis of the brain or indicate the presence of disease of the cerebral arteries. Their subjects are liable to hemorrhage in the brain and all its consequences. The changes which have been described are of the greatest importance, according to Foster Moore, if they are concerned with the secondary and tertiary branches of the retinal artery.

W. L. Benedict seeks to discriminate between the retinal lesions of hypertension arteriosclerosis and acute and chronic nephritis.

H. P. Wagener has recently offered the following classification: Secondary, senile, primary, and consecutive retinal angiosclerosis. The symptoms ascribed to each type are briefly as follows: In the senile type there are reduction of the apparent caliber of arteries and veins, but with regular lumina, no arteriovenous compression, but pallor of the disk, sclerosis of the choroidal vessels, and often spots of guttate choroiditis (page 395). Aged persons may have, however, practically normal eyes, or only a slight lessening in the size of the vessels; also they may develop a true arteriosclerosis. In primary arteriosclerosis, that is, endovasculitis, there are exaggeration of the retinal arterial reflex and beading, irregularities in the caliber of the retinal arteries, arteriovenous compression, tortuous terminal arterioles, areas of obliteration of arteries, new vessel formation, perivasculitis, and dusky congestion of the disk. (See also paragraphs 3 and 4, page 519.) In consecutive arteriosclerosis, due to general vascular hypertension, there are exaggerated retinal arterial reflexes, generalized reduction in the caliber of the retinal arteries, irregularities of their lumina, venous engorgement and *straight arteriovenous compression, i. e.*, the course of the vein is not altered, but beading of the vessels, new vessels, perivasculitis, and spots of arterial obliteration are absent. Implanted upon a fundus of this character there may be scattered white patches, hemorrhages, constituting the type Benedict has named "retinitis of hypertension plus nephritis," and if the hypertension is malignant, hemorrhages and edema are marked.

In the secondary arteriosclerotic group are included those vascular changes seen after subsidence of renal retinitis and other varieties of retinitis and neuroretinitis and of papilledema which have been elsewhere referred to.

P. C. Bardsley endeavors to differentiate ophthalmoscopically between the signs of arteriosclerosis and those due simply to increased blood-pressure. In cases of simple increased blood-pressure the vessels have the appearance of uniform distention, the light streak is larger than normal, but the silver-wire appearance in the arteries is absent; indentation of the veins occurs if the arteries are very tight.

In *retinal angiosclerosis* the disease is probably primarily in the intima, according to Coats, and in arteritis new tissue forms which encroaches on the lumen of the vessel, sometimes suggesting a hyaline change. According to this observer, irregularities of vessel caliber are due to endothelial proliferation, and silver-wire arteries to fibrosis. Endothelial proliferation of the main artery depends upon a circulating toxin.

Angioid Streaks in the Retina (*Retinal Pigment Striæ*).—These occur as dark, reddish-brown, sometimes almost black striæ lying beneath the retinal vessels. They give the impression of a system of obliterated vessels. According to Ward Holden, they are caused by

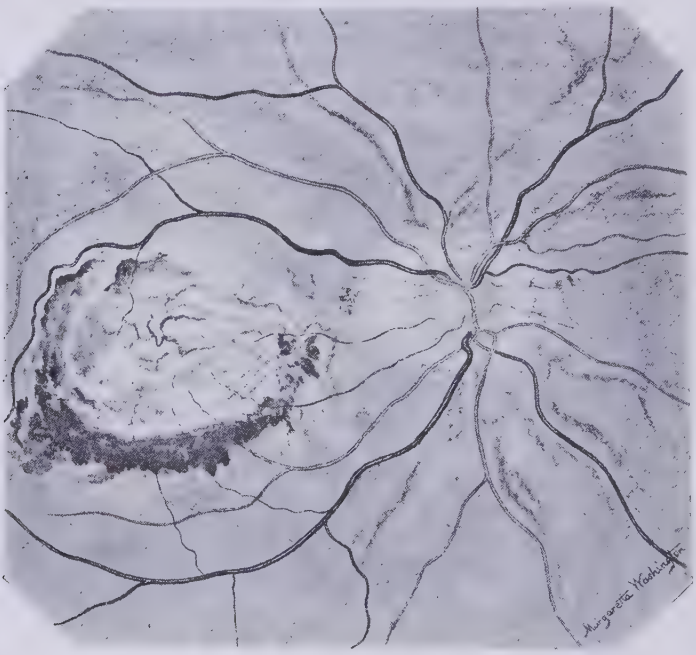


FIG. 235.—Angioid streaks in the retina; large central semicircular hemorrhage.

metamorphosis of hemorrhages diffused in a linear manner through the deep layers of the retina. Lister thinks they represent newly formed vessels which have penetrated inflamed tissue, and along which pigment deposits and other exudations are arranged. W. Zentmayer is inclined to regard the streaks as pigmented vessels which are either of inflammatory or congenital origin. Coats suggested that the streaks arise along vessels, but that these vessels belong to the choroid rather than the retina, and Treacher Collins attributes them to hematoidin and hemosiderin in subchoroidal and intrachoroidal hemorrhages. They may occur in several members of the same family, as was the case in a series examined and studied by the author.

Exudative Retinitis (*Retinitis Hæmorrhagica Externa; Massive Retinal Exudation* [Coats]).—In this form of retinal disease, particularly well described and studied by George Coats, the most conspicuous feature is a large, prominent yellowish-white circumscribed lesion, or smaller areas of yellow or white exudations lying beneath the retinal vessels. Coats investigated several varieties of the affection, namely, those without gross vascular disease, those with extensive vascular changes, and those with arteriovenous communication. Later he removed the last-named variety from his classification of massive retinal exudation. Of insidious onset and slow progress, the disease most often attacks young persons (average age about nineteen), and is more common among males than females. The patients are usually

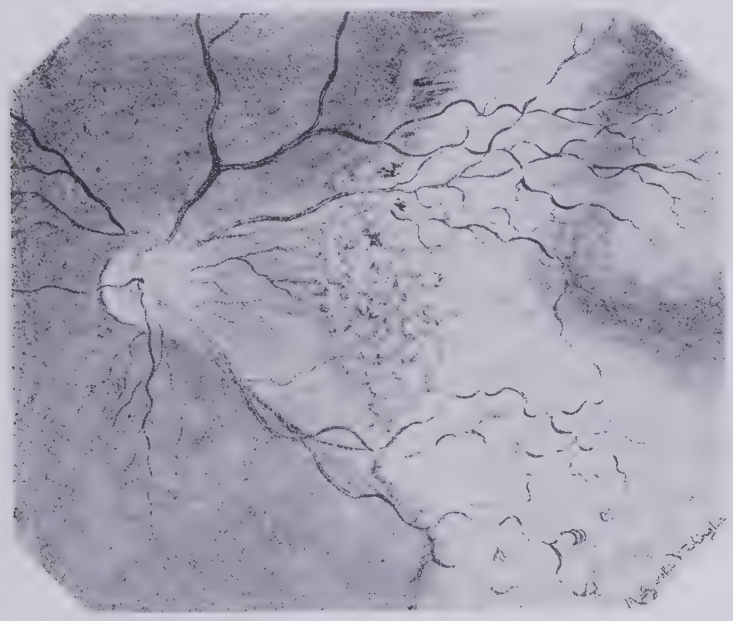


Fig. 236.—Massive retinal exudation. Left eye of a girl aged nineteen.

in good health (anemia may be present), and their clinical and family histories do not yield information as to the etiologic factor. In late stages of the disease detachment of the retina, cataract, iritis, and glaucoma may develop. The affection depends, as Coats has shown, upon hemorrhages in the inter-retinal layers. A slow organization takes place with formation of cicatricial tissue masses. At first the choroid remains free from pathologic alterations, but there are borderline cases of the disease characterized by involvement of the choroid as well as the retina in the form of thickening and round-cell infiltration. Exudative retinitis is probably the result of local vascular disease; ophthalmoscopically it has most often been mistaken for tuberculous choroiditis. From the clinical standpoint this disease has been

studied in this country by Friedenwald, Zentmayer, Jervey, the author, and other observers. Verhoeff has made one pathologic examination.

Aneurysms.—Aneurysm of the central retinal artery is an extreme rarity. It has been seen as a spindle-shaped sac, pulsating synchronously with the heart. Miliary aneurysms, usually spindle shaped, have been noted on the small arterial twigs in elderly persons, and may be looked upon as significant of a similar condition of the vessels in other organs, especially the brain. They may be associated with angiosclerosis, cardiac disease, and nephritis. The student should not mistake varicosities in the veins for aneurysms. Arteriovenous aneurysm of the retina has been described. *Retinitis with miliary aneurysms* is a term descriptive of a lesion consisting in an extensive infiltration of the retina with multiple miliary aneurysms, particularly investigated by Leber and, although resembling one of the varieties of exudative retinitis with vascular changes (Coats), has been placed in a separate class. All of the cases (about 15 in number) have occurred in young males. No satisfactory cause for this condition has been discovered.

Angiomatosis of the Retina.—This rare disease has been observed by Fuchs, Goldzieher, Darier, and a few other observers, but its first accurate description is by E. von Hippel and it is sometimes called *von Hippel's disease*. According to him, the most prominent ophthalmoscopic appearances are red, spheric formations, with enormous dilatation and tortuosity of one or more arteries and the accompanying veins, both sets of vessels having the same color. Something over 30 cases are on record; tuberculosis is suggested as a cause. Anatomic examination reveals diseased retinal vessels, destruction of the nervous elements, proliferation of the glia, and organization of subretinal hemorrhage (von Hippel). According to Meller, primarily there is increase of the neuroglia with secondary vascular changes. The prognosis as to vision is fatal; blindness from secondary glaucoma results.

Obstruction of the Central Artery of the Retina, Including Embolism and Thrombosis.—An embolus may lodge in the central artery of the retina. Lesions of this distinctive character have been observed by Harms, Schweigger, Manz, Marple, Coats, and others. The symptoms recorded in the following paragraphs may also be caused by thrombosis, by obliterating endarteritis, and by vascular spasm. Leber, however, was of the opinion that embolism is much more frequently the cause of the obstruction than the reports of investigators would indicate.

Symptoms.—The main branches of the *artery* are thin, and can be followed only a short distance over the edge of the papilla into the retina, and there is a diminution in the number of ramifications. The *veins* are also contracted, and they may present ampulliform broadening, alternate contractions and swellings, and especially a contraction at the disk, succeeded by broadening in the periphery, where they assume almost their natural breadth. Pressure from before backward,

so as to increase the intra-ocular tension, causes a regular current to flow through the vessels. This consists of broken cylinders of blood, separated by clear spaces, which move sluggishly along. In the veins, without such pressure, and, it may be, directly after the accident, an *intermittent blood-stream* is often visible. The appearance is not unlike that produced when air is allowed to mix with a fluid in a tube. Occasionally a few hemorrhages are seen along the course of the vessels.

The *papilla* assumes a pallid, grayish-white appearance, owing to the lack of blood in its capillaries. An *opacity in the retina* develops, grayish-white or fog-like in appearance, sometimes permitting the reddish tint of the normal eye-ground to shine through it, and sometimes being so opaque that it is quite milk-like in its density. This opacity is apt to select the neighborhood of the papilla and of the macular region, a space between the two areas sometimes being free, although gradually they meet. The opacity is visible within a few hours after the accident, or it may be delayed for a day or two. The author has watched it form within twenty minutes after sudden stoppage of the central retinal circulation. It is due, according to Coats, not to edema (the usual explanation), but to an *ischemic necrosis*.

Characteristic of sudden obstruction of the arterial circulation is the formation in the macula lutea (corresponding to the position of the fovea) of a central red spot, which resembles a round hemorrhage in the midst of the milky-white area. It is known as the *cherry-red spot* of the macula lutea, and is caused by the red color of the choroid appearing through the much-thinned retina, and changes in the pigment epithelium. As a rare phenomenon, at least in the dark-skinned races, the usual cherry-red spot has been replaced by a coal-black one. The spot appears at the same time the opacity in the macula lutea develops. It is less likely to form where there is a stoppage of a branch of the retinal artery instead of one of the main trunks.

In the course of several weeks there is a gradual disappearance of the retinal opacity, the optic disk undergoes atrophy, and the retinal vessels are shrunken or even converted into white cords; if there have been hemorrhages, spots of degeneration appear at their positions, and not infrequently cholesterin crystals and pigment markings may be seen around the disk and in the macula lutea.

Instead of the *main trunk*, a *branch* may be obstructed, and this obstruction is sometimes visible to the ophthalmoscope as a yellowish body, but, more frequently, at one point of the artery there is a swelling, while beyond it the vessel is obliterated or its caliber is much reduced.

Vision is lost with characteristic suddenness. Usually preceding the blindness there is temporary obscuration of vision, or a little headache and giddiness, with flashes of light, representing a species of aura. Periods of temporary blindness lasting from a few minutes to one-half hour, during several years (in one of the author's cases twelve years), may precede the ultimate obstruction of the artery. In obstruction of a branch, on the other hand, there may be very good acuteness of vision. The presence of a *cilioretinal vessel* may be the means of

preserving vision. A rare condition is obstruction of a cilioretinal artery (Hirsch, Zentmayer, F. Krauss).

The *field of vision* varies according to the extent of the blocking of the circulation. In cases where the obstruction is complete, even light-perception is absent. If only a branch has been occluded, the function of that portion of the retina which receives its blood-supply from this source will be paralyzed, and the corresponding area of the field will be darkened. The presence of a cilioretinal vessel permits, as a rule, an oval portion of the field of vision to remain in the neighborhood of the fixation-point, but, according to C. F. Clark, the evidence is not sufficient to warrant the conclusion that such a vessel is the means

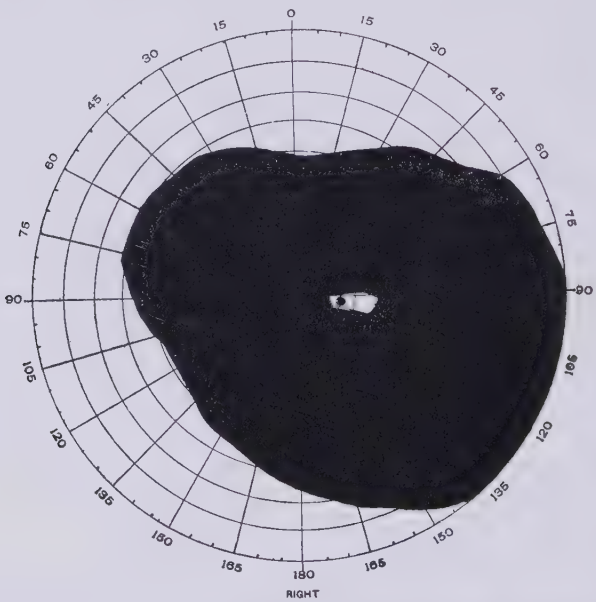


FIG. 237.—Small visual field representing area of functionating retina surrounding blind-spot.

of preserving the integrity of the papillomacular region of the retina. Even if the main stem of the artery is obstructed, a small portion of the field may be preserved on the temporal side, corresponding to an area on the nasal side of the fovea in the region of the blind-spot (Fig. 237). An uncommon effect is a central scotoma, which may be due to obstruction of the macular arteries; the scotoma may also have a paracentral situation.

The intra-ocular tension is sometimes raised, sometimes lowered, and sometimes unaffected. The pupil may be large and irresponsive to light if the case is one of complete stoppage of the central artery.[1] As complications there may be retinal hemorrhages, perhaps due to

[1] The symptoms which have been described refer to typical cases; a variety of exceptions occurs.

associated venous thrombosis (Leber), secondary glaucoma, and rarely an iritis.

Causes.—The most frequent causes of obstruction of the central artery of the retina are valvular disease of the heart, especially if complicated by a fresh endocarditis, and general arterial sclerosis, aneurysm of the aorta or of the carotid, Bright's disease, and pregnancy; in a few instances it has been noted in chorea; also in association with anemia, menstrual derangements, recurring epistaxis, and diabetes. Thrombotic obstruction depends upon endarterial changes, and alteration of the composition of the blood and its coagulability. Stoppage of the central artery may occur at almost any age of life, and has been recorded from the fifteenth to the eightieth year. The accident usually is unilateral, simultaneous obstruction of the central artery of each eye being very rare; occasionally both eyes are affected, with a definite interval between the attacks.[1]

Blindness with ophthalmoscopic appearances exactly similar to those caused by obstruction of the central artery has followed subcutaneous injections of paraffin in the nasal region and injections of bismuth paste into the accessory sinuses. Fat embolism of the artery and its temporary obstruction by a bubble of air have been reported.

Diagnosis.—The ophthalmoscopic picture just detailed indicates that a block in the central retinal circulation has occurred, due usually to one or other of the causes already named. Similar appearances have been ascribed to hemorrhage into the sheath of the optic nerve, to spasm of the muscular wall of the central artery, and to thrombosis of the central vein so situated that it presses upon and occludes the lumen of the artery lying beside it. Schweigger taught that emptiness of the arteries was an important sign of true embolism.

Some cases of obstruction of the central retinal circulation appear to be due to collapse of the arterial walls, so that they come in contact (Hoppe). In these circumstances recovery may occur spontaneously or be brought about by treatment. While it is true that "there is at present no proof that obstruction may be caused by *spasm* apart from endarteritis" (Coats), the effect of an apparent spasm of the retinal arteries from the clinical standpoint must be conceded. The author has examined Harbridge's patient, and watched complete collapse of the retinal arteries, followed in four minutes by the restoration of their caliber. Spasm may be due to toxic agents, even to tobacco (Ormond). *Intermittent closing of the retinal arteries*, apparently due to vessel cramp, has been many times observed; for example, in association with angiosclerosis, Raynaud's disease, epilepsy, and migraine (see also page 481).

Prognosis.—This is exceedingly unfavorable, and in most instances blindness is the result. Even where temporary improvement occurs,

[1] In a certain number of cases (30 per cent. according to A. Knapp), although all the ordinary ophthalmoscopic appearances of embolism of the central artery of the retina have been present, it has been impossible to assign a cause. This is particularly true in cases occurring in young persons, especially young women.

subsequent atrophy of the nerve is likely to ensue. In obstruction of a branch the prognosis is more favorable, and, as has been stated, normal central vision may be present. The presence of a cilioretinal vessel improves the prognosis. According to Swanzy, in cases of bilateral obstruction, unless vision is lost simultaneously, a fair amount of vision may be restored. Glaucoma, as before noted, may follow stoppage of the central retinal artery.

Treatment.—In the hope of restoring the circulation by reducing the intra-ocular tension, sclerotomy, iridectomy, and repeated paracentesis of the anterior chamber have been practised, and successes have been reported.

Massage of the eyeball has been recommended, and in some cases has been followed by good results. It should be given a faithful trial; the author can confirm its value. Inhalations of nitrite of amyl should be given. If massage fails, operation may be performed (paracentesis or iridectomy, preferably the former).

Thrombosis of the Central Vein.—In thrombosis of the central vein the obstruction practically always occurs at the lamina cribrosa and may be associated with narrowing of central artery.

Symptoms.—The ophthalmoscopic signs of this condition may be similar to those described under Hemorrhagic Retinitis, of which it may be a cause (see page 492). Several grades of this condition have been recorded. The ophthalmoscope may reveal tortuosity of the vessels, engorgement of veins, and normal or contracted caliber of the arteries, venous pulse, and interrupted venous circulation and extensive retinal hemorrhages; or there may be complete obscuration of the disk, which is hidden by infiltrated retina, and surrounded by large flame-shaped and sometimes sheet-like hemorrhages, which extend widely over the fundus. Yellowish-white spots or areas may appear between the hemorrhages. Thrombosis of the central veins and retinal angiosclerosis may be coincident and obstruction (partial) of the central artery or one of its branches may also occur. Instead of the main trunk, one of its branches may be thrombosed, and the ophthalmoscopic appearances confined to the area which it drains (Fig. 238). Vision is much reduced; a central scotoma may be present. A "hole in the macula" has been observed (Williamson). Sometimes the peripheral field is uncontracted; in other cases pronounced defects are discoverable indicating implication of a retinal artery.

Causes.—It occurs in cardiac disease, arteriosclerosis, nephritis, diabetes, rheumatism, gout, and influenza; occasionally in chlorotic anemia and infectious diseases. Sometimes a history of antecedent phlebitis of the extremities is obtained, as in several female patients under the author's care. In a certain number of cases no definite somatic malady can be found—it is a local disease.

Prognosis.—If the thrombosis is in the central vein, hope of restoration of vision practically must be abandoned; in obstruction of a branch the prognosis is more favorable. A *secondary glaucoma*, sometimes developing acutely, is not an uncommon complication, due to ob-

struction of the filtering area by an albuminous exudation. This complication must always be feared. Foster Moore has made an interesting and important observation, namely, that although the intra-ocular tension of the affected eye is usually less than that of the unaffected eye, should acute glaucoma subsequently develop, the intra-ocular tension of the unaffected eye is either equal to or greater than that of the affected eye. Evidently systematic tonometric measurements are indicated. If glaucoma develops, treatment is unsatisfactory and usually the eye passes into a stage of so-called absolute glaucoma (page 423). Retinal vein thrombosis in a definite number of cases appears to be an indication of future cerebral hemorrhage. *Iritis* with the formation of fibrovascular membrane has been observed.

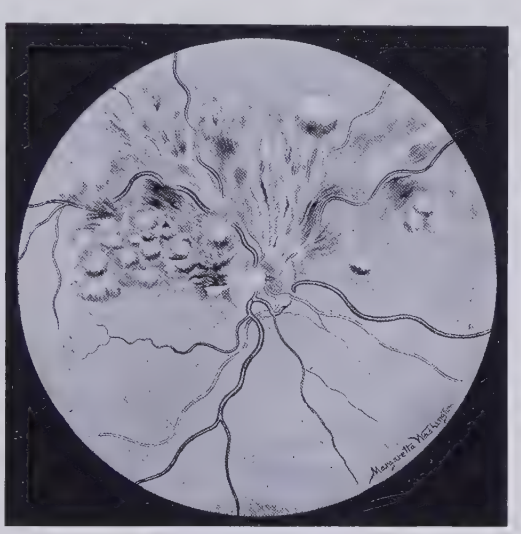


Fig. 238.—Thrombosis of a retinal vein.

Treatment.—This depends upon the general condition, and patients with central vein thrombosis should be thoroughly studied from all standpoints and treatment directed according to the results of the examination. Focal infection in the teeth and tonsils should be eliminated. Other things being equal, iodids and syrup of hydriodic acid may be administered. Diaphoresis may be tried; mydriatics should not be used lest they cause glaucoma; indeed, it is proper to use miotics as part of the routine treatment; dionin is of service.

Traumatisms of the Retina.—Under this general term may be included traumatic anesthesia, traumatic amblyopia, traumatic perforations of the macula lutea, detachment, and rupture. There are no characteristic symptoms common to all varieties, but pain and disturbance of vision, in part due to the direct injury and in part to a transient astigmatism, are likely to be present.

1. *Traumatic anesthesia of the retina* is the name which was proposed

by Leber to describe effects of a blow upon the eye without discoverable ophthalmoscopic changes, but with considerable defect in vision and contraction of the visual field—results, moreover, which may remain unchanged for a long time, or, indeed, never entirely pass away.

The *treatment* is rest and the use of strychnin internally, or by hypodermic medication.

2. *Traumatic amblyopia* (*commotio retinæ; edema of the retina*) is a condition also arising from an injury, especially a blow from a ball, cork, or similar body, and is attended by the following symptoms: Hyperemia of the globe marking the position of contact of the missile; clear media; and gray opalescence of the retina, especially in the

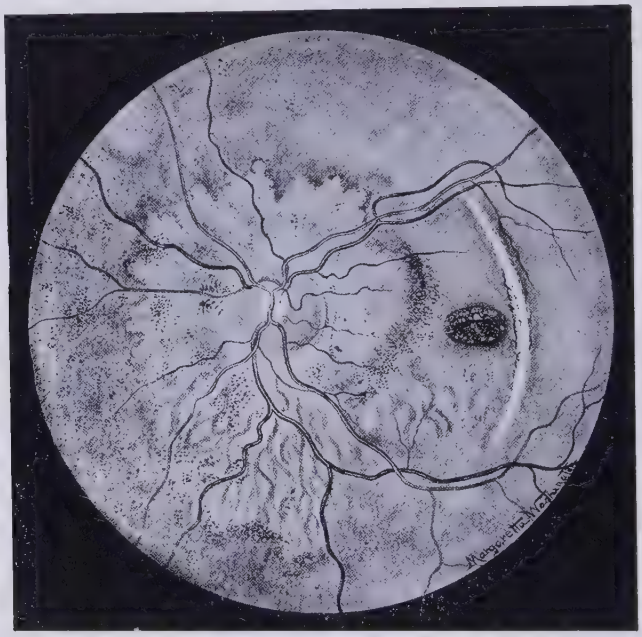


Fig. 239.—Hole in macula and rupture of choroid after a blow on eye (patient in the University Hospital).

macular region, but also around the papilla, which may be somewhat hyperemic. If the retina under the point of contact is visible, this also may exhibit a white infiltration. In addition, several pale yellowish spots and, occasionally, small hemorrhages may be present. The vessels are unchanged or, in some instances, are contracted (arteries) or distended (veins) and pass *over* the gray area. A central scotoma may exist.

An interesting complication is the development of a transitory astigmatism, which helps to reduce the visual acuteness.

The gray infiltration forms quickly and is also absorbed with rapidity, usually having subsided at the end of two or three days, although

the visual defect may last for longer periods. Decided retinochoroiditis, the result of concussion, may occur, and this fact should be remembered in investigating cases of choroidal disease of long standing. According to Fuchs, changes in the macula after contusion may be due to inflammatory edema as the result of a low-grade inflammation of the ciliary region. The lesions produced by concussion of the eyeball during warfare, as noted so frequently during the past war, occur as the result of concussion at a distance (explosion of a shell), by transmission of concussion through the bony facial structures, or by impact of a missile.

The *commotio retinæ* produced by such war injuries differs somewhat from the type common in civilian life described above, in the

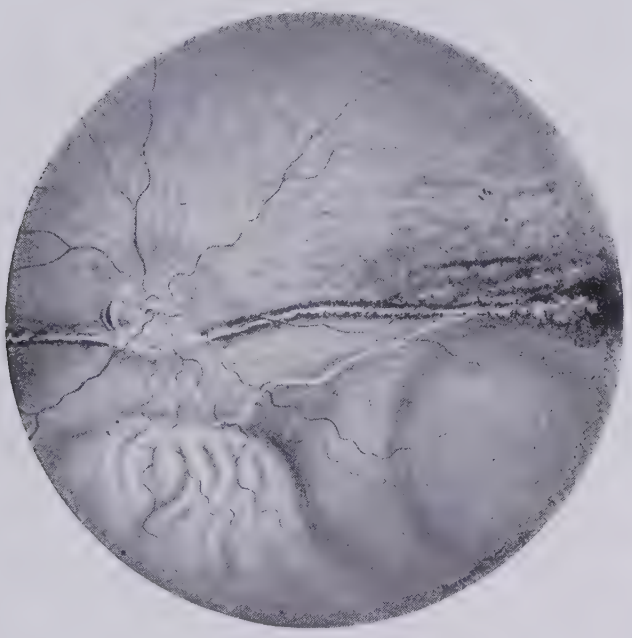


Fig. 240.—Traumatic detachment of the retina and chorioretinal rupture.

presence of more numerous hemorrhages, the yellow tint of the so-called retinal haze, its longer duration, and more circumscribed character.

An interesting but evanescent picture has been observed and particularly described by Sir William Lister in the later stages of commotio retinæ, after disappearance of the haze; namely, peculiar striæ in the vicinity of the macula, almost certainly due to the wrinkling of the swollen retinal layers.

In the "grossly concussioned fundus" in the early stages there are widespread clouds of hemorrhage, many gleaming and glistening white particles, which gradually fade, become converted into fibrous

tissue, and represent patches of coagulation necrosis from rupture of retinal and choroidal vessels (Lister).

The *treatment* of ordinary commotio retinæ consists in keeping the pupil dilated with atropin and covering the injured eye with a shade or dark glass, all use of the uninjured organ being forbidden.

3. *Traumatic Perforations of the Macula Lutea.*—Haab drew attention to the fact that a contusion or concussion injury of the eye may cause a round hole in the macula about half the size of the surface of the optic disk, surrounded by a gray ring. The bottom of the hole is of reddish color, with a stippling of white and red and is below the level of the surrounding retina. F. M. Ogilvie is responsible for the apt term which describes this lesion—*hole in the macula*. In some cases the retina is detached; in others it is not. A central scotoma may exist. Occasionally the lesion is bilateral due to a simultaneous injury of each eye (T. M. Li). Holes in the retina in regions other than the macula may be produced by trauma and choroiditis (Lister). Other signs of injury may be present in the eye-ground—for example, rupture of the choroid (Fig. 187). This "hole" in the macula is produced by an edema of the retina at the posterior pole, followed by cystic degeneration (Coats). Some hours must elapse after the injury before the lesion is evident. Traumatic "holes in the macula" are not of very uncommon occurrence in civilian practice; numbers of them were observed during the past war, sometimes as the sole lesion, more often in association with extensive fundus lesions, particularly ruptures of the choroid and retina.

4. *Detachment of the retina after injury* has been mentioned (see page 513).

5. *Rupture of the Retina.*—Rupture uncomplicated by choroidal fissure the result of injury is a rare accident, and might be recognized by observing the frayed edges of the tear and seeing the exposed choroidal tissue. Long records a case of this character following a fall upon the back of the head.

Purtscher has described a fundus lesion after fracture and traumatisms of the skull, to which he gives the name *angiopathica traumatica retinæ*. The ophthalmoscopic picture consists in the presence of shining white patches, chiefly associated with the veins; hemorrhages may also occur. These patches are supposed to be due to cerebrospinal fluid which has been forced into the retinal perivascular lymph-spaces. To this condition Körber applies the term *lymphorrhagia of the fundus.*

Retinal Changes from the Effect of Sunlight (*Solar Retinitis*) **and Electric Light** (*Electric Retinitis*).—It has been experimentally proved that retinal changes can be produced in animals' eyes by concentrating upon them the rays of the sun. Clinically, analogous disturbances have been found in the human retina after exposure to intense light, most frequently in those who, with unprotected eyes, have watched an eclipse of the sun (*eclipse blindness*). Similar conditions are caused by intense electric light, especially among those engaged in electric welding.

The **symptoms** are: Persistence of an after-image or, later, a dark spot in the field of vision (positive scotoma); distortion of objects, and evidences of slight retinitis or retinochoroiditis in the macular region. Thus, there may be a maroon-colored area with a central gray patch, and numerous faintly marked yellowish-white dots. A cherry-red spot has been detected by Ischreyt and a definite "hole in the macula" has been described.

Decided improvement is not infrequent, but complete recovery is exceptional (Mackay); hence prognosis must be guarded. The central scotoma may be permanent (Duane); ring-shaped scotoma has also been observed. Degeneration of the papillomacular bundle may occur (E. T. Collins).

The **treatment** is that suited to retinochoroiditis. The preventive treatment consists in wearing suitable colored glasses—yellow glass or a combination of blue and red, or, as in Sheffield, several layers of ruby glass.

Glioma of the Retina (*Retinocytoma* [Mawas]).—This is a malignant growth of the retina, and is a soft, vascular tumor, made up of small round, deeply staining cells, many of them containing long protoplasmic processes. They form thick mantles of well-preserved cells around the thickened blood-vessels, the cells between the mantles staining poorly and undergoing calcareous degeneration. In many of these neoplasms peculiar *rosettes* have been observed, first described by Flexner and well studied by Wintersteiner, which are composed of elements resembling the rod and cone visual cells, and for these growths the name *neuroepithelioma* has been suggested. According to Alt the rosette formation is due to the growing of tumor-cells around a tissue enclosure, and not to rudimentary rods and cones, and Ginsberg believes they correspond to cells of the rudimentary retina, which are not differentiated into spongioblasts and neuroblasts. Glioma usually arises from the inner retinal layers; less frequently from the outer retinal layer. According to Leber, it may develop from various layers not only in different eyes, but in the same eye (Parsons). Exactly how glioma originates is not certainly known, but probably in fetal retinal cells. Its pathologic histology in no way resembles that of glioma of the brain, Hence the term "glioma" is unfortuntae, but it is so firmly associated with this tumor of the retina that it is difficult to find a satisfactory substitute.

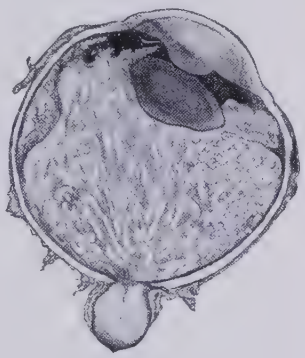


FIG. 241.—Glioma of retina (patient in the University Hospital).

According to the direction which the growth takes, it has been described by systematic writers as *glioma endophytum* and *glioma exophytum.* In the former the vitreous chamber is occupied by the growth; in the latter, it lies between the retina and choroid.

The tumor is usually of a light gray or grayish-red color. It is subject to various degenerative changes—fatty, cheesy, and calcareous—and tends, on the one hand, to invade the orbit, involve the optic nerve, and travel by the way of its sheath to the brain, and, on the other, to pass forward, bursting through the sclera and cornea. Recurrence *in loco* after extirpation may occur, and metastases, especially in the cranial and facial bones and the brain, may take place. They also occur, according to F. M. Wilson and E. S. Thomson, in the lymph-glands, parotid, liver, ovaries, kidneys, spleen, lungs, and spine. Glioma



FIG. 242.—Recurrence of glioma, forming the so-called fungus hæmatodes (from a patient in the Philadelphia General Hospital under the care of Dr. Hearn). Photograph by Dr. Pfahler.

may cause changes in and invade tissues of the eye other than the retina: the vitreous, choroid, iris, ciliary body, and anterior chamber.

Like sarcoma of the choroid, glioma passes through several stages. In the first, there are no signs of irritation, the media are clear, the pupil is dilated, and often the growth produces a whitish reflection which has given rise to the designation *amaurotic cat's eye* (see also page 399). As the disease progresses symptoms of irritation and increase in the size and tension of the globe become manifest, and the process begins to involve the optic nerve. Finally, the tumor bursts from its bounds, perforates the globe at its corneoscleral junction, grows rapidly, involving the orbit and neighboring temporal regions,

and presents a huge vascular mass, to which, in former times, the name *fungus hæmatodes* was applied.

Glioma of the retina is probably always congenital. It may apparently occur as late as the fifteenth year (one case in a girl of twenty [C. Maghy]), but in such circumstances it is probable that the growth has been present, but has remained quiescent. In Berrisford's report on 42 cases the tumor was observed at birth in 3, within the first year in 9, during the second year in 6, during the third year in 3, during the fourth year in 4, during the fifth year in 3, and during the sixth year in 2. It is not a common affection. Hereditary glioma has been reported (Hill Griffith, Traquair). Several members of the same family may be affected. One or both eyes may be involved (bilateral in about 20 per cent. of the cases [A. Knapp]).

Diagnosis.—The following conditions, according to E. T. Collins, may be mistaken for glioma: Persistence of the posterior part of the fetal fibrovascular sheath of the lens; masses of tubercle in the choroid; inflammatory or purulent effusion into the vitreous following retinitis or cyclitis, usually with detachment of the retina (see also Pseudoglioma, page 470). Circinate retinitis (white degeneration of the retina), according to de Wecker, has been mistaken for glioma. The author and E. A. Shumway have recorded a case of detachment of the retina with extensive dropsical degeneration of the rod and cone visual cells which exactly simulated glioma. In glioma the anterior chamber is uniformly shallow; in inflammatory exudations into the vitreous the chamber is deepened at its periphery (retraction of iris) and shallow at its center (bulging of pupillary border). Synechiæ are occasionally present in glioma. Tension is usually increased in glioma, but may be minus; rarely the tension is elevated in pseudoglioma. In case of doubt the eye should be enucleated. Glioma may occur in a shrunken eye (about 21 cases on record [Berrisford]).

Sarcoma of the choroid is differentiated from glioma by the fact that the former usually occurs at a later period of life, and that in the earlier stages of each affection the ophthalmoscopic findings are different. In glioma the tumor is seen to *involve* the retinal structure, which does not, as in sarcoma, merely act as a covering to the growth. Unlike sarcoma, glioma is never pigmented.

Prognosis.—This is unfavorable, and if the disease has involved the optic nerve or burst from its bounds, it is fatal. Although so-called spontaneous cure has been observed (Lindenfield), as an almost invariable rule unmolested glioma causes death. Numbers of recoveries after proper enucleation are on record, and an opinion must be based on the extent of the disease, the condition of the optic nerve being the most important element in the prognosis. Even enucleation of both eyes, not at the same operation, has been performed with success. According to Hirschberg, a favorable prognosis may be given if the tumor has not passed the limits of the retina, and if the time elapsing since the first appearance of the growth has not exceeded ten weeks. Recurrence is rare after three years of immunity. In a number of fatal

cases which have been analyzed (Lawford, Collins) the optic nerve was unaffected in only 4. In unfavorable circumstances recurrence takes place in the orbit, with extension to the brain, and, more rarely, metastasis to a distant organ.

Treatment.—Thorough enucleation, with division of the optic nerve as far back as possible, is the only surgical treatment. The optic nerve should be immediately examined; if it contains glioma cells the contents of the orbit should be eviscerated. In several instances both eyes have been removed, and recovery after such procedure has been recorded—for example, by Simeon Snell. Recently *x*-rays and radium have been used in the treatment of glioma, and there is evidence that the growth may be checked by this means. It is important that the first

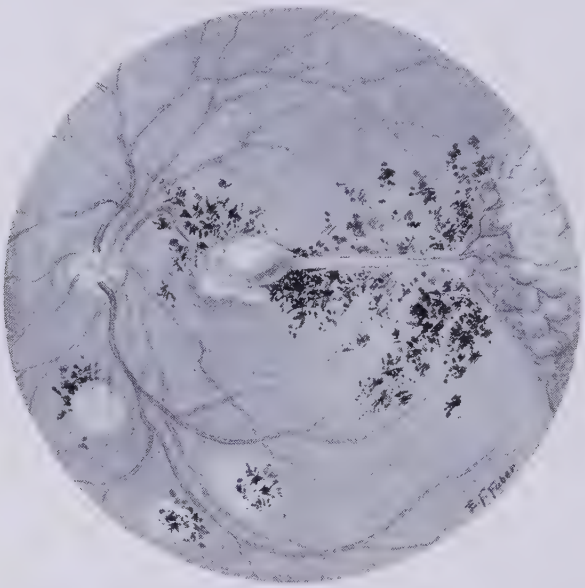


FIG. 243.—Congenital multilocular cysts in relation with the retina, and associated with quiescent pigmentary retinochoroiditis (from a patient at Fort Oglethorpe, Ga.)

dosage should be as large as safety permits. However, even with shrinking of the globe metastasis may occur. Verhoeff achieved satisfactory results by employing *x*-ray therapy. In 17 cases of glioma reported in literature thus treated, analyzed by J. Jacoby, only 3 were "successful." After the enucleation of one eye the other eye should be most carefully examined; glioma in its earliest stages may thus be detected. Following the enucleation radiation of the socket is advisable.

Subretinal Cysticercus.—This, like the presence of the same parasite in the vitreous, is exceedingly uncommon in this country (see also page 477).

Cysts of the Retina.—Small cysts or cystic spaces due, probably, to arteriosclerosis of the retinal capillaries are not infrequently found in the eyes of old persons just behind the ora serrata (E. T. Collins).

Cysts are also found in the detached retinas of blind eyes, where they may be multiple. They may commence in the degenerated retina, or be due to the adhesion of folds of this membrane (Parsons). Sometimes they are detected by means of the ophthalmoscope. In a patient under the author's care a large retinal cyst springs from the upper part of an inferior detachment of the retina, leans over, and partially obscures the disk. G. S. Derby describes a similar cyst. Meyer Wiener and the author observed, clinically, in a young soldier ophthalmoscopic appearances which were reported as congenital cysts in relation with the retina (Fig. 243).

Symmetric Changes at the Macula Lutea in Infancy (*Tay-Sachs' Disease*).—This disease, which occurs almost always in children of Hebrew parentage and begins from the third to the sixth month of life, is also known as "amaurotic family idiocy" (a name given by B. Sachs); it was first described by Warren Tay, and consists of a grayish-white zone about the size of the papilla in each macular region, with a brownish or cherry-red spot in the center, closely resembling the appearances seen in obstruction of the central artery. At first the remainder of the fundus is normal, but later the optic disk undergoes atrophy. The general clinical signs are: muscular enfeeblement, apathy, mental weakness, paralysis, and gradual loss of sight. Death occurs in from one to two years. The autopsies show a change in the pyramidal cells of the cortex and degeneration of the cord. According to Sachs, this is an arrest of development. Ward Holden, by Nissl's method, has shown that there is degeneration of the retinal ganglion cells, and his results have been confirmed by Shumway and others. Verhoeff attributes the dark spot in the macula to contrast; no edema or hole was found in this region. The ocular manifestation is merely, as Sachs insists, one symptom of a family (several members may be affected) disease. Syphilis is not a cause of the disease.

Family Cerebral Degeneration with Macular Changes.—This affection, first described by R. D. Batten and named by Oatman "maculocerebral degeneration" (familial), presents itself in two forms: (*a*) The maculocerebral type, both retina and brain being affected, and (*b*) the macular type, the lesions being confined to the retina. It develops at a later age than family amaurotic idiocy, and is not race selective. In the first type the disease begins usually about the sixth year of life and is characterized by macular pigmentation, progressive blindness, progressive paralysis, and dementia (Batten and Mayou). In the second type the period of development is delayed and begins about the thirteenth year, and the central nervous system is not affected. Such cases have been described by Stargardt as "progressive family degeneration in the macular region." Brown Pusey's investigations of this type of the disease suggest to him it may be an expression of early arteriosclerosis. These and allied lesions have been discussed by Gifford under the title of "Juvenile Types of Amaurotic Family Idiocy," and by Darier as "Progressive Familial Macular Degeneration."

Leber has described a variety of *tapetoretinal degeneration*, bilateral and occurring usually in several members of the same family. There is a *central* form with changes (white spots and pigment grains) confined to the maculopapillary region, and a *diffuse* form with widespread pigment changes. According to the age at which the disease develops it is denominated *infantile* or *juvenile*. This affection is a frequent cause of congenital blindness. Sometimes the central type is delayed until middle life. The affection may be associated with cerebral degeneration resulting in idiocy, but must not be confounded with amaurotic family idiocy (page 537). This ocular disorder probably

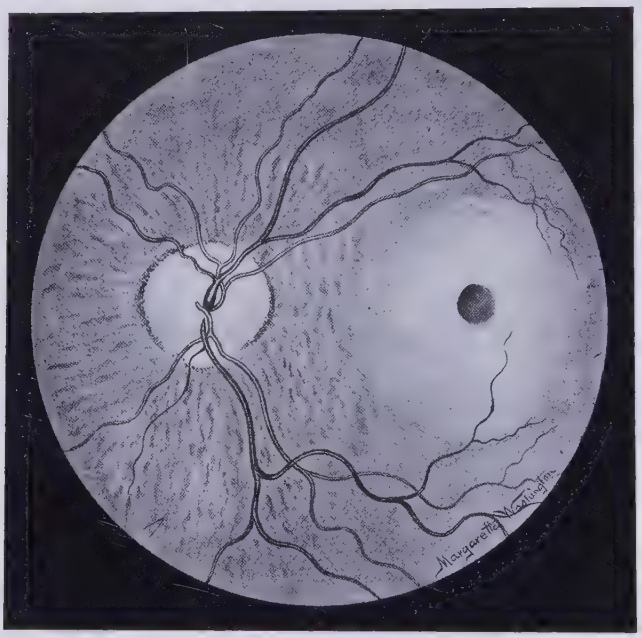


FIG. 244.—Changes at the macula lutea in amaurotic family idiocy (from a patient in the University Hospital).

belongs to, and should be classified with, the group of pigmentary degenerations of the retina (page 508).

Senile Macular Atrophy of the Retina.—Various types of central or macular retinochoroiditis and degeneration have already been described, and it is not uncommon to find in the eyes of old persons, in the macular regions, areas of yellowish-white spots interspersed with pigment dots and small hemorrhages, or irregular areas of erosion which may go on to atrophy of the elements and pigment heaping. Haab has called attention to a pure retinal senile affection consisting of yellowish-red or whitish, or else darkly pigmented spots, the rest of the eye-ground being normal, and Harms, by microscopic investigation, demonstrated that the lesions depend upon an atrophy and dis-

appearance of the involved tissues, affecting chiefly the neuro-epithelial layer. The pigment epithelium is much altered, but the choroid is practically not affected. Senile macular changes seem to exercise a deterrant effect on the development of cataract.

Retinitis Atrophicans Centralis.—Kuhnt, Haab, and the author have described a macular affection, which in all particulars in its ophthalmoscopic appearances resembles the lesion known as traumatic perforation, or "hole" at the macula (see page 532). It may be caused by retinal vascular disease, and a similar appearance may arise as the result of a non-traumatic iridocyclitis or from a toxin. Indeed, Fuchs

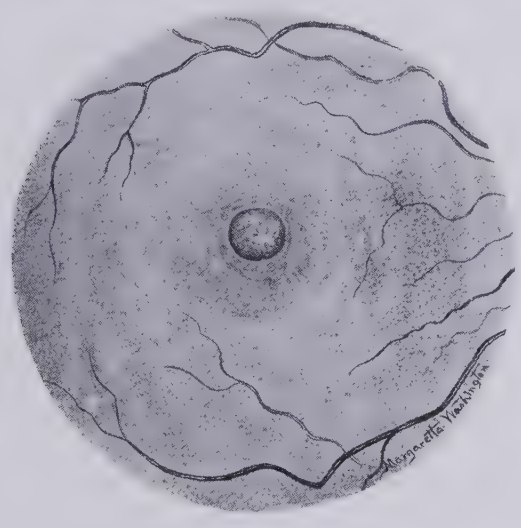


FIG. 245.—"Hole in the macula"; no trauma; marked general arteriosclerosis.

has shown that *hole-formation in the macula* may occur in a variety of conditions; for example, iridochoroiditis, neuroretinitis, retinal detachment, and retinitis pigmentosa.

Treatment.—These macular changes do not respond to treatment, in the sense that any remedy is available which can dissipate the lesions. Nevertheless, patients thus affected should not be dismissed as "hopeless." The persistent use of certain alterative remedies, the best being Donovan's solution, and of dionin, is advisable; subconjunctival injections of physiologic salt solution, not too frequently, may be tried; sinusoidal galvanism has been recommended. Sources of focal infection, especially in the mouth, bladder and prostate, should be eliminated.

CHAPTER XV

DISEASES OF THE OPTIC NERVE

Congenital Anomalies.—Medullated Nerve-fibers.—In the normal eye the fibers of the optic nerve cease to be invested with a medullary sheath at the lamina cribrosa, and consequently the axis-cylinders, which are distributed to the retina, are transparent. As an anomalous condition, sometimes bilateral, but more frequently only in one eye, the medullary sheaths reappear at the upper or lower margin of the disk as a dull or glistening bluish-white patch, which extends for a variable distance out into the retina, and ends in a somewhat feathery or fan-shaped margin. Usually the retinal vessels are hidden by the patch, but reappear again on its distal side.

This plaque may be a single one above or below, or it may appear both above and below the disk, more rarely on the nasal side, and very exceptionally upon the temporal margin. The size varies from a small expansion to a huge sweep of white tissue, continuous above and below with margins of disk, and taking somewhat the general direction of the vessels, which are wholly or in part concealed. Medullated nerve-fibers of the retina at a considerable distance from the disk have been reported by Randall, Nettleship, Arnold Lawson, and other observers, and B. T. Lang has observed them passing outward toward the temporal periphery encircling the macula up to the horizontal raphé. In their usual situation they are interpreted in the visual field by an enlargement of the blind-spot, which, however, according to H. Gradle's investigation, seldom corresponds to the size and shape of the ophthalmoscopic picture.

Coloboma of the Sheath of the Optic Nerve.—This congenital anomaly is characterized by an apparent augmentation of the surface of the disk and an excavation of the papilla backward and downward. The periphery is usually bounded by pigment massing. There is an unequal division of the retinal vessels, which are first seen as they bend over the margin of the excavation. It is a rare anomaly, and has been mistaken for posterior staphyloma. It depends upon imperfect closure of the fetal fissure.

Irregularities in the Disk.—Instead of its usual round or oval shape, the disk may be markedly irregular in outline, one side being occasionally at an apparently lower level than the other, or it may present a gibbous appearance. *Congenital pigmentation of the optic nerve-fibers*, most intense in the position of the physiologic excavation, has occasionally been described; the pigment may exist as a small spot or cover an area one-fifth of the size of the disk. Sometimes it exactly surrounds the exit of a vessel.

When the nerve-head fails to fit the choroidal aperture accurately, a space is sometimes formed, usually crescentic, known as a "cone" or "conus." This generally is seen at the outer side of the papilla, but also inward, below, and very rarely above (see also page 141). It should not be confused with the atrophy of the choroid seen in myopic eyes, to which the name *posterior staphyloma* is given (see page 143), nor with the crescents of choroiditis seen in astigmatic and stretching eyes, in which the scleral ring broadens out into a semi-atrophic area of disturbed choroid, usually bounded by an irregular pigment line, and most commonly developed at the temporal side of the disk. *Con-*

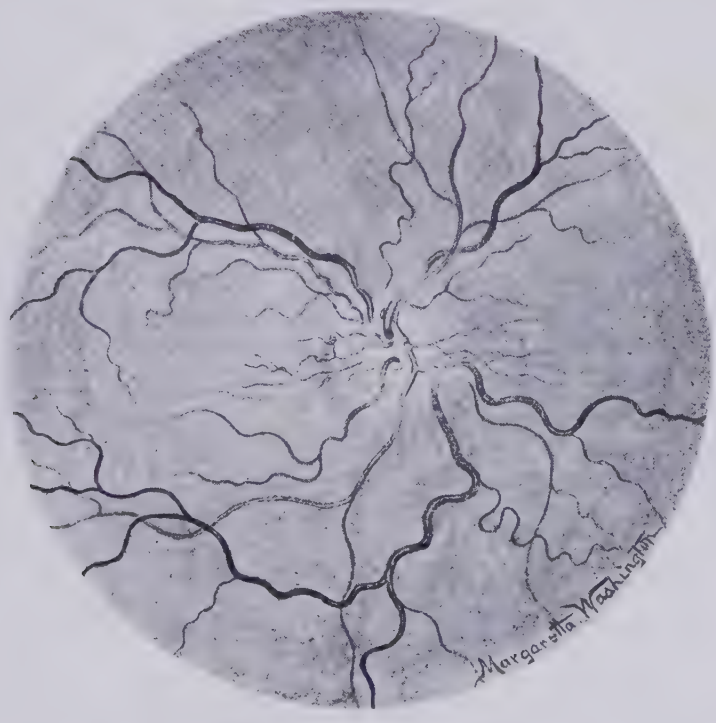


Fig. 246.—Spurious optic neuritis. High degree of hyperopic astigmatism (from a patient in the University Hospital).

genital inferior crescent or *conus* is caused, according to Elschnig, by a pulling away of the choroid from the disk margin. The affected eyes are usually astigmatic.

Shreds of Tissue on the Disk.—These appear as glistening white patches of tissue, sometimes almost transparent, at other times thicker and more opaque, either completely or partially hiding the vessels. Occasionally there is a white membrane more or less completely covering the disk.

Such appearances probably represent remains of the hyaloid artery or of its adventitious coat.

Hyperemia of the Nerve-head (*Congestion of the Disk*).—The color of the intra-ocular end of the optic nerve varies considerably, and it is not accurate to describe a nerve-head as congested if it simply is redder than usual.

As Gowers pointed out, the term *simple congestion* is suitable when the papilla presents a dull red or brick-dust hue, which shades almost imperceptibly, through a blurred margin, into the general red color of the fundus; when it is more marked in one eye than in the other, the latter serving as a picture for comparison; when at some antecedent examination the same optic disk has presented a more natural color; and when its borders are obscured, but not hidden.

In other circumstances—and the appearance is a frequent one—the surface of the nerve is covered by a semitransparent or edematous layer, is unduly injected, and its margins, especially the nasal ones, are veiled by striations composed of fine grayish lines and minute capillaries ordinarily not visible. The perivascular lymph-sheaths at the same time are unduly prominent in the form of white lines along the central vessels, especially the veins. This appearance has received the name "hyperopic disk," and has also been called "spurious optic neuritis" (Spicer) and "pseudoneuritis" (Stephenson). Some cases of pseudoneuritis are congenital in origin. (See Fig. 246.)

Causes.—(*a*) Refractive error, especially hyperopia and hyperopic astigmatism. (*b*) Prolonged exposure to glare and heat. (*c*) Certain toxic agents presently to be described, and inflammation of the iris, usually of the syphilitic type. (*d*) Certain disorders of the brain, especially various types of chronic insanity. Focal brain lesion—for example, cerebral embolism—may be associated with hyperemia of the nerve-head. Pathologic disk-hyperemia at one time reported as frequent in the second stages of syphilis, while it undoubtedly occurs and may yield, though stubbornly, to antiluetic treatment, is much less frequent than the earlier records indicate. It is at times difficult to decide between hyperemia and a beginning neuritis.

Treatment.—This depends entirely upon the cause. Refractive error should be corrected. Constitutional measures are required if there is reason to believe that some general cause is at work.

Anemia of the Nerve-head.—This is not a disease peculiar to the optic nerve, but, like retinal anemia, occurs as part of a general anemia, or because of obstruction in the central vessels—for example, in embolism.

It is often most difficult to interpret the significance of pallor of the papilla. It requires more than ophthalmoscopic examination to decide whether or not a pallid disk is pathologic; careful visual field and light-sense testing are necessary.

1. **Intra-ocular Optic Nerve Inflammation and Edema.**—For convenience these conditions may be described as (*a*) *intra-ocular optic neuritis*, or *peripheral optic neuritis*, and (*b*) *engorgement-edema of the papilla*, or *choked disk*.

If the lesions consist of hyperemia and moderate swelling of the

PLATE VI.



The fundus of the right eye of a patient with tumor of the brain, showing choked disc.

nerve-head and no unusual overfilling of the veins, and of an exudation which produces discoloration and opacity of the papilla, so that its margins and surface are obscured or hidden, and the whole process is not strictly limited to the disk, but passes into the retina immediately surrounding it, *descending neuritis* is often employed as a descriptive term.

If the lesions are chiefly confined to the nerve-head itself, and there are great swelling and engorgement, suggesting mechanical compression, marked distention of the retinal veins, and hemorrhages in and near the edematous papilla, the term *choked disk*, or *papilledema* (Elschnig, Parsons) is used to describe the condition.

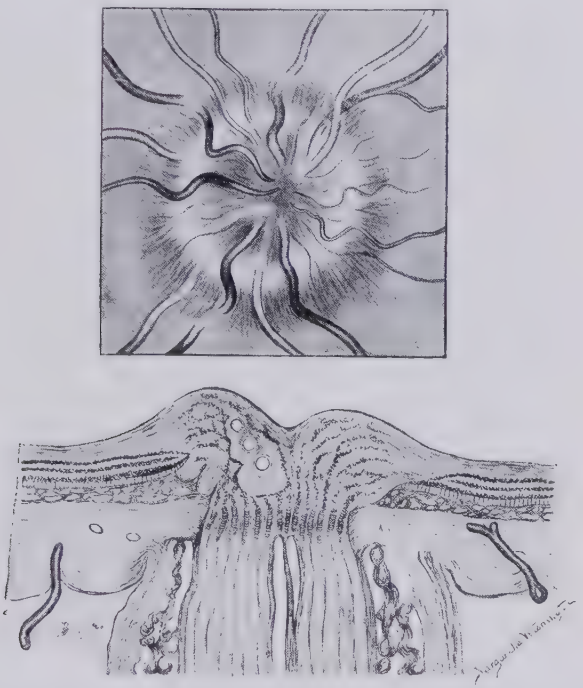


Fig. 247.—Ophthalmoscopic picture of papilledema and semidiagrammatic representation of a longitudinal section of the nerve-head.

If the retina is extensively involved, with hemorrhages along the vessels, spots of degeneration, sometimes collected in a star-shaped figure analogous to that seen in renal retinitis, the term *neuroretinitis* is frequently utilized.

To avoid this confusion of names, Leber proposed the general term *papillitis*, and if it is understood to refer only to those types evidently of inflammatory origin, the word is suitable.

Symptoms.—In general terms, the symptoms which follow belong to the conditions now under consideration, but vary in their intensity or elaboration chiefly in so far as the swelling of the papilla is concerned,

according as neuritis (optic neuritis) or engorgement edema (choked disk, papilledema) is present.

1. *Changes in the Nerve-head.*—(*a*) There are increased redness of the disk and obscuration of its borders, followed by swelling of the papilla, loss of the light-spot, and complete hiding of the margins, the center usually remaining more red than the periphery, which has a grayish tint and shades gradually into the surrounding retina. The swelling may increase, assume a mound shape of mixed grayish color, and finally the form of the disk is lost, and its position can be inferred only by the convergence of the vessels. White spots and patches are often seen at the elevation sometimes covering the retinal vessel. The height of the swelling is estimated by means of the ophthalmoscope (see table, page 117).

2. *Changes in the Vessels.*—The *arteries*, smaller than normal, pursue a moderately straight course and are difficult of recognition, being



Fig. 248.—Papilledema; section of nerve-head (from the William F. Norris Collection, University of Pennsylvania).

partly concealed by the swelling. Occasionally spontaneous pulsation is visible. The *veins* are dark in color, distended and tortuous, and pass along the slope of the elevation, often dipping into the infiltrated tissue. The light-streak is not lost, at least not where the vessel is clearly visible. The tortuosity of the vessels is sometimes remarkable, and has been compared to the writhing snakes in the *Medusa-head.* The point of emergence and convergence of the vessels may be hidden by the infiltration, so that the center of the swelling seems somewhat destitute of vessels. In some instances thickening of the adventitia of the vessels gives rise to the appearance of white lines along their sides.

3. *Hemorrhages.*—In many cases hemorrhages are found upon the swollen papilla or in its immediate neighborhood. They are in the form of narrow, flame-shaped extravasations if they lie in the fiber-layer, but may also assume other shapes if situated in a deeper plane.

The number varies from a single hemorrhage to so many that the swollen nerve-head assumes a *hemorrhagic* form, or the surrounding retina may be freely occupied by elongated or other shaped patches of blood. Usually optic neuritis and choked disk are bilateral, although it is not uncommon to find the process more advanced in one eye than the other. Occasionally the condition is confined to one eye and the other remains unaffected.

In addition to the ophthalmoscopic changes just detailed, the following points deserve notice:

1. *Vision* in optic neuritis and in choked disk may be defective or it may be entirely unaffected; hence the mere presence of good central sight should never be considered cause to omit ophthalmoscopic examination. Usually the vision of one eye is more affected than its fellow. Impairment of sight may come on rapidly or slowly. Occasionally vision is lost with great suddenness, but this is rare. Photometric examination usually reveals disturbance of the *light-sense* (see page 70); the light-difference (L. D.) is defective (A. S. Percival).

2. The *field of vision* presents for consideration its periphery, which may at first be unaffected and later show irregular and concentric contraction; an increase in the size of the normal blind-spot, which becomes correspondingly greater as the swelling increases; and the formation of a scotoma due to involvement of the axial fibers and occasionally simulating the Bjerrum type (V. Szily); the absence of half of the visual field (hemianopsia) if the intracranial mischief which may have been the cause of the neuritis or choked disk is so situated as to produce this phenomenon. Cushing and Bordley have described reversal of the color lines as it occurs in hysteria in the visual field in association with increased intracranial tension, with and without choked disk, as well as blue-blind areas, which disappeared after the restoration of intracranial tension to normal by operation. Although reversal of the color lines in these circumstances is demonstrable, it must not be regarded as a safe indication of the existence of increased intracranial tension, and Cushing has ceased to regard this symptom as one having diagnostic importance.

3. *External Appearances.*—There are no changes in the exterior of the eye indicative of swelling or inflammation in the nerve-head. There are no characteristic *pupillary* phenomena. The pupil may be moderately dilated, but, as Kampherstein has shown, in the majority of cases of choked disk its reaction is normal. If blindness is complete, the iris usually is immobile. Normal reaction, however, has been noted even in the presence of complete blindness (Kampherstein). (See also page 67).

Diagnosis.—The diagnosis of optic neuritis and of choked disk depends upon a direct ophthalmoscopic examination of the inflamed or edematous disk. The method of determining the height of the elevation has been explained.

The student should not mistake the slightly prominent disks that are occasionally seen in hyperopia for beginning papillitis or papille-

dema. If the condition is due to intracranial disease the disk edges are more blurred than in *pseudoneuritis*, the physiologic pit is contracted or filled in, the veins are darker and usually more tortuous, and a careful examination of the size and shape of the blind-spot, of the light-sense, and of the visual field should, in most instances, establish the diagnosis.

The distinction between choked disk and optic neuritis is often difficult. Obviously, it cannot entirely be made according to the degree of swelling which is present (2 D. or more characterizing choked disk, according to Uhthoff). The ophthalmoscopic evidence in each case has been referred to (page 543). Papillitis associated with various types of retinitis has elsewhere been described. Recently the Gullstrand slit lamp has been utilized in the early and differential diagnosis of optic neuritis and choked disk, especially by Koeppe. Using special apparatus and illumination he finds that the anterior vitreous is normal in the *early* stages of choked disk, while in optic neuritis, whitish sharply defined lymphocyte flakes are visible. In *incipient choked disk* the papillary structure is visible, but in inflammatory optic neuritis it is clouded or obliterated. Many other differential points are developed for which the original must be consulted.[1]

The average swelling of papilledema in cerebral tumors is, according to the author's and Holloway's measurement, 4.57 D. It varies from 3 to 9 or 10 D. Particularly satisfactory studies of the various stages and types of choked disk and papillitis can be made with the large Gullstrand ophthalmoscope (page 99).

The course of the choked disk is a variable one. Occasionally swelling of the intra-ocular end of the nerve develops with great rapidity; in other instances it is slow in its course and lasts for months and even years, with progressive failure of vision. While in a certain sense the various stages into which systematic writers have divided choked disk (papilledema) and optic neuritis are artificial, they are convenient for descriptive purposes. The following, referring especially to choked disk, are those, somewhat modified, which were recorded by Marcus Gunn:

1. Increased redness of the disk, with blurring of its upper and lower margins, with a gradual progression of the blurring to the nasal edges, while the temporal margin is still visible, represents the first stage.

2. Increased edema of the nerve-head, beginning filling in of the physiologic pit, involvement of the temporal margin of the disk, with a tendency of the edema to spread into the surrounding retinal area, and uneven distention and darkening of the retinal veins represent the second stage.

3. Decided increase of edema, elevation and size of the nerve-head, with vascular striation of the swollen tissue and striæ of edema in the form of lines in the swollen retina between the disk and macula, marked distention of the retinal veins and retinal hemorrhages represent the third stage.

[1] Graefe, Arch. f. Ophth., 1922, clx, 454.

4. Increase in the prominence of the disk, which assumes a mound shape and begins to lose its reddish color and juicy appearance and to become opaque, exudation in and on the swollen disk and surrounding retina, elaboration of the retinal hemorrhages in size and number represent the fourth stage.

5. Decided subsidence of the vascularity of the papilledema and increasing pallor, with or without sinking of its prominence, apparently contraction of the retinal arteries and thickening of their perivascular lymph-sheaths, spots of degeneration in the retina, especially in the macula, represent the fifth stage, which passes into the final stage of so-called *papillitic atrophy.*

As the last stage is ushered in the borders of the disk begin to be visible, usually first upon the temporal side, until finally all margins again are apparent, at first a little mellowed, while the center is still covered by the remnant of the inflammatory tissue. Finally, the edges of the disk are clear, its color is white and atrophic, and its center becomes apparent. Both sets of vessels are contracted, and may be streaked along their sides with whitish tissue. Areas of retinochoroiditis and elevated patches of degeneration, marking spots of former hemorrhages, are often apparent. Second attacks of neuritis and choking of the disk may occur, as in a case observed by the author and A. G. Thomson. A choked disk may be implanted on an atrophic nerve-head.

In addition to the swelling of the disk, there may be marked *edema of the retina* and lines of edema in the macular region, forming the so-called *macular figure,* or *macular fan* (Paton), not unlike the appearance which is so striking in certain types of renal retinitis (see page 493). It occurs in a fair percentage of cases of cerebral and cerebellar tumors (fully 15 per cent. according to Paton's figures), and may reach a height equal to or greater than that of the choked disk. In addition to these areas of retinal edema, there may develop in the macular region yellowish-white and degeneration spots, intermixed with hemorrhages.

The **prognosis** of optic neuritis and choked disk depends upon the cause and the duration of the process. If, for example, syphilis is the active agent, there is reason to hope that suitable treatment may be effective. If the focus of disease, for instance, in the accessory sinuses, can be removed, vision may be saved and edema and inflammation will subside. If the papilledema depends upon increased intracranial tension, and this is relieved by decompressive trephining, or by a radical operation with removal of the growth, and the disk changes have not passed beyond the third stage, the prognosis as to sight is favorable. Untreated choked disk, or optic neuritis, almost always produces great depreciation of vision or blindness; very exceptionally the original disease continues, but the neuritis subsides (Oppenheim).

Causes.—The most frequent cause of choked disk is *tumor of the brain,* inasmuch as it occurs in fully 80 per cent. of the cases. Usually the intracranial neoplasm must have existed for some time and the increased intracranial tension must have lasted for a definite period

before the engorgement edema develops. The period from the beginning of choked disk to the height of its swelling in some instances comprises only a few weeks; in others, months and even years may elapse before the disk edema appears. It is not possible to determine with certainty from the stage of the papilledema what the duration of the cerebral lesion is, but if choked disks arise with suddenness and the edema rapidly increases, the indications are that an increase in intracranial pressure has occurred, either because the growth itself has gained in volume or because hemorrhage has occurred in or around it.

Tumors of the corpora quadrigemina give the highest percentage of choked disk, and next tumors of the parieto-occipital region and of the cerebellum, which yield an almost identical percentage (tables of John E. Weeks and J. M. Martin). Tumors of the basal ganglia are usually associated with papilledema. Choked disk, if it does not fail entirely as a symptom of tumor of the pons, of the medulla, and of the corpus callosum, is apt to be late in its development, and, to a certain extent, this lateness of development applies to tumors of the frontal and parietal convolutions. It is probable that pontine tumors give rise to choked disk only if they also involve some neighboring structure, and, according to Paton's researches, the bulk of cases of brain tumor without choked disk are those of pontine and subcortical origin, but if the subcortical growths spread to the base, the disk changes appear. Tumors of the cerebellum are prone to cause a more intense form of choked disk, with rapid depreciation of vision, than cerebral neoplasms, and the same intensity of the process is, according to some authors, evident in morbid growths of the midbrain and thalamus, while it is less pronounced in subcortical, parietal, and frontal lobe tumors. Whether the refraction of the eye has any influence on the development of choked disk is undecided. That myopia seems to have a deterrent effect has been asserted (Marcus Gunn, the author); its influence in this respect is doubted by other observers (Paton, Parsons, Bordley, and Cushing). The development of choked disk does not necessarily depend upon the size, situation, or structure of the intracranial neoplasm, and all types of morbid growths may originate papilledema—fibroma, sarcoma, glioma, carcinoma, solitary tubercle, and gumma.

It also appears with echinococcus cysts, epidural and subdural clots, intracranial trauma, abscess of the brain, and middle-ear disease, when this has extended to the cerebrum. Von Hippel describes a form of optic neuritis with affections of the ear which may exist without disturbing sight and remain even after operation has removed its apparent cause. (See also page 551.) *Acute blindness* with normal fundus due to the pressure of internal hydrocephalus caused by brain abscess has been described (Pagenstecher).

Injuries to the skull (blows, fractures, etc.) may be followed by rapid disk-edema (beginning choked disk), and in all such cases repeated ophthalmoscopic examinations are required. During the past war a large opportunity arose for demonstrating the value of such examina-

tions, no matter, as Greenwood has said, whether the cranial injuries were simple concussions, furrow wounds, fractures, or penetrating wounds. Finding disk changes and noting their character (choked disk or papillitis) often furnished the indication for operation. Almost always swelling of the papilla appearing soon after a cranial injury indicates a developing choked disk. Disk changes at a later period may fully develop into papilledema (choked disk) and be due, for example, to a cyst, or papillitis (optic neuritis) may be dependent on a meningitis or brain abscess. Bilateral choked disks arising weeks or months after cranial injury are of grave import in that they indicate a serious intracranial lesion which had escaped notice.

Of the four varieties of meningitis—simple, tuberculous, traumatic, and cerebrospinal—tuberculous disease of the brain is the most frequent cause of optic neuritis, the percentage varying from 76 to 81 per cent. The appearances of the disk may be those which have been described in connection with *descending neuritis* (see page 543). In the case of direct pressure upon the tracts and chiasm, the swollen papilla has a peculiar grayish-white color, without much vascularity, and a similar appearance is sometimes caused by tumors of the cerebellum. In epidemic cerebrospinal meningitis, optic neuritis, that is, a descending neuritis, or choked disk from distention of the third ventricle, may develop. These optic nerve changes are not frequent according to Uhthoff (17 per cent.). The author's experience, especially during the war, would indicate a higher percentage. Both choked disk and optic neuritis occur with otitic meningitis.

Other intracranial causes are softening of the brain, hemorrhagic pachymeningitis, cerebritis, hemorrhage, thrombosis of the cavernous sinus, hydrocephalus, aneurysm, and disorders of the pituitary body and extrasella growths.

Choked disk is common in cerebral syphilis, which, next after tumor, is said to be its most frequent cause (Uhthoff). The cerebral affections in this respect have been classified by this author as follows: Gumma of the brain and its membranes; gummatous basilar meningitis; syphilitic lesions of the cerebral vessels and their sequels; internal hydrocephalus of syphilitic origin. Igersheimer states that a *typical* choked disk may probably develop in association with cerebral lues, even though anatomic demonstration is lacking.

Occasionally general paresis, epilepsy, and disseminated sclerosis are accompanied by optic neuritis and by choked disk. Papillitis may precede, accompany, or follow myelitis (ophthalmoneuromyelitis).

In addition to the intracranial causes of papillitis, this phenomenon may arise from a general infection. To this form Uhthoff gives the name *infectious optic neuritis*. According to this observer, it should be differentiated from those cases which are caused by orbital, intraocular, or intracranial lesions, and may be caused by any of the following diseases placed in order of their frequency: Influenza, syphilis, rheumatism, malaria, typhus fever, measles, whooping-cough, diphtheria, polyneuritis, smallpox, beriberi, erysipelas, scarlet fever,

tuberculosis, typhoid fever, gonorrhea, and relapsing fever. The neuritis may manifest itself as a papillitis or as a retrobulbar neuritis, and Uhthoff thinks that the optic nerve conditions are most apt to arise during the stage of convalescence and are probably due to the action of toxins, and not directly to the micro-organisms. The meningitis which may complicate various infectious diseases may be the agency in the development of papillitis and choked disk. In children with congenital syphilis, even when only a few weeks old, optic neuritis is not uncommon, according to Mohr. Optic neuritis may also be caused by toxic agents, for example, by lead, atoxyl, Filix mas, and alcohol; by anemia, both when this is an essential process and when it is caused by excessive hemorrhage; by disturbances of menstruation,

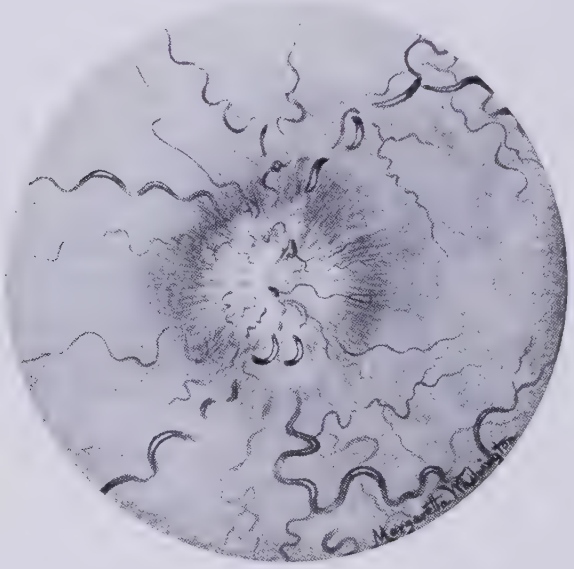


Fig. 249.—Fundus of the right eye of a patient with tumor of the brain and choked disk; swelling 6 D. (Service of Dr. Edward Martin in University Hospital.)

by lactation, by exposure to cold, by myxedema, by sunstroke, and by injuries. An association of chlorosis, choked disk, and abducens palsy has been observed, attributed by Meller to thrombosis in the region of the cavernous sinus. Marked disk-edema (choked disk) may complicate chlorotic anemia.

Metastatic optic neuritis has been recorded as occurring in sepsis (Axenfeld, von Michel). Optic neuritis may be associated with diseases and injuries of the anterior part of the eye (iritis, iridocyclitis). Under these conditions vitreous opacities may also be present.

Optic neuritis followed by atrophy may arise in association with *deformities of the skull*, and, according to H. Friedenwald's analysis, the patients for the most part have had oxycephalic or steeple-shaped skulls ("tower skulls"). Blindness without changes in the intra-ocular

end of the optic nerve due to cranial deformity have been reported. Papillitis occasionally occurs as a congenital affection in several members of the same family and sometimes appears without evident cause.

Choked disk and optic neuritis may depend upon disease of the orbital region—inflammation of its contained tissues, tumors, caries, and periostitis, especially around the optic foramen, upon purulent disease of the antrum of Highmore and the frontal sinus, and morbid processes of the upper posterior portion of the nose and of the sphenoid and ethmoid bone (*sinusitis*). In most of these instances, unless both orbits or the sinuses are affected, the papillitis is unilateral. Optic neuritis may be due to dental disease and to other focal infections, for instance, in the tonsils. The relation of focal infections in the teeth and



Fig. 250.—Fundus of the right eye of the same patient shown in Fig. 249 one month after cerebral decompression.

tonsils and of paranasal sinus disease to papillitis is an important one to which much attention has been directed in recent years. Purulent middle-ear disease and mastoid infection by virtue of intracranial complications may be accompanied by papillitis; should this arise, the indication for operation is evident. Optic neuritis with cavernous sinus thrombosis is elsewhere discussed (page 664).

A rare form of optic neuritis is that described in association with persistent dropping of a watery fluid from the nose. Headache, vomiting, unconsciousness, and delirium are present. The fluid has been believed to be identical with the cerebrospinal fluid (Leber), or to be due to nasal disease in the form of small polypi (Nettleship and Priestley Smith). Internal hydrocephalus was present in some of the patients.

Treatment.—This depends upon the cause of the condition. In all syphilitic cases rapid mercurialization should be tried, followed later by the iodids. Salvarsan and neosalvarsan have been effectively employed. Orbital and sinus diseases and focal infections indicate appropriate surgical measures.

Since Sir Victor Horsley's announcement, more than forty years ago, that the release of intracranial tension arrests and cures optic neuritis (choked disk), numerous operations have been performed, with satisfactory results. In a certain number of cases of brain tumor the growth can be removed by a radical operation, but even if it is inoperable, as it frequently is, or cannot be localized, a palliative operation, that is, *cerebral decompression*, should be performed in order to save sight, and the earlier it is done, the better the results will be—*i. e.*, operation should be undertaken before the third stage of the disk change is reached. Usually, after technically correct operations (and in all pretentorial tumors temporal decompression is the operation of choice, and in all subtentorial lesions a suboccipital decompression is indicated [C. H. Frazier]), the choked disk begins to subside from the third to the tenth day, and the subsidence is complete at the end of six weeks. The same operation is urged by Cushing in choked disk caused by cerebral edema, infections, and intracranial hemorrhage. Choked disk has also been advantageously treated by lumbar puncture, and W. G. Spiller and the author have published some very successful results secured with the help of this procedure; only a small quantity of fluid (5 c.c.) should be withdrawn at a time. This caution lessens the danger of lumbar puncture in the presence of brain tumor. *Puncture of the corpus callosum* and drainage of the ventricle have been utilized to reduce intracranial pressure and thus relieve papilledema.

Significance of Choked Disk.—Double choked disk is highly significant of intracranial disease, especially tumor or basilar meningitis. Indeed, it is the most important general symptom of this condition, but it is not a pathognomonic sign. The other causes of optic neuritis and choked disk which have been mentioned must be excluded, and care must be taken not to mistake the macular figure (see page 547) for a renal retinitis. Although the presence of choked disk is so highly significant of cerebral tumor, of itself it possesses no distinct localizing importance. Usually papilledema is bilateral, but in a certain number of instances it is unilateral, and frequently there is an excess of choking in one eye as compared with the other. Whether this is a sure indication that the tumor is on the same side as the choked disk or the excess of edema is undecided. Horsley believed that choked disk tends to develop earlier and to be more marked in the eye corresponding to the side on which the tumor grows, but Paton doubts if reliance can be placed on this sign. In the author's and Holloway's investigations in the majority of cases the greater swelling was on the same side as the tumor; but there were many cases in which this rule did not hold good. The development of choked disk does not depend upon the size nor on the type of the tumor, although according to some authorities disk

changes are most frequently absent in tuberculous growths and most frequently present with sarcoma, glioma, and cysts. According to Walter R. Parker, choked disk caused by increased intracranial tension appears first in the eye with the lesser intra-ocular tension, as measured with the Schiötz tonometer. As this author himself points out, this observation requires confirmation.

Pathogenesis of Papillitis and Choked Disk.—As is well known, von Graefe at one time sharply distinguished between *descending neuritis* and *choked disk* (*Stauungs-papille*).

If, for example, in meningitis the sheaths of the optic nerve, which are continued over it as prolongations of the corresponding brain membranes, participate in the inflammation, as they undoubtedly may, there is at first, as Greeff points out, a *perineuritis*, which extends by way of the connective-tissue septa to the trunk of the nerve. The evidences of this inflammation, soon visible to the ophthalmoscope, present the appearances of a moderate, that is, not engorged, intra-ocular optic neuritis, and the whole process is a *descending neuritis*.

If, on the other hand, the state of the nerve-head indicates engorgement, edema, and mechanical obstruction, and the evidences of these conditions are visible to the ophthalmoscope in the appearances already described (see page 543), the process is a *choked disk* or *papilledema*.

Inasmuch as ophthalmoscopically it is frequently difficult to distinguish a neuritis from a beginning choked disk, and as the conditions may be mixed, Hughlings Jackson expressed the opinion that there is one kind of optic neuritis from intracranial disease which may manifest itself under different appearances, sometimes with and sometimes without "swelling of the disk." It would seem, however, that it is still proper to maintain, within the limits described, the distinction to which reference has been made.

Numerous theories have been propounded to explain the *pathogenesis of choked disk*. Von Graefe believed that choked disk was due to a venous stasis occasioned by obstruction to the return of venous blood from the cavernous sinus. This theory ceased to be tenable when Sesemann demonstrated the anastomosis between the ophthalmic and the anterior facial veins. Parinaud taught that choked disk is due to extension of the interstitial edema of the brain tissue through the optic nerve to its intra-ocular end, a theory to which Sourdille subscribes; and Kampherstein believes that often it can be explained only by a preceding edema of the brain, extending through the optic nerve to the lamina cribrosa and thus causing choking of the nerve-head.

The *inflammatory* or *toxin theory*, with various modifications, assumes, as Leber suggested and Deutschmann afterward endeavored experimentally to show, that so-called papillitis is not a product of edema, but an inflammatory affection, the fluid which distends the sheath of the nerve possessing an irritative quality; or, in other words, that the subarachnoid fluid is infected by products from the intracranial disease or lesion which is the prime cause of the trouble.

The *mechanical (lymph-space) theory* of Schmidt-Rimpler, ascribed to the dropsy of the intersheath space of the optic nerve, which is caused by the increased subarachnoid fluid being forced into this situation under the influence of elevated intracranial pressure, a mechanical or compressing action, or to the fluid which found its way into the lymphatic spaces of the optic nerve, an action causing edema, congestion, and later inflammation.

Although in the investigation of choked disk from the experimental standpoint the results of various observers have not always been in accord, on the whole it has been demonstrated, as W. R. Parker has well shown, that by artificially increasing intracranial pressure choked disk may be produced. Choked disk, according to Schieck, is due to lymph stasis, the cerebrospinal fluid passing by way of the perivascular lymph-sheaths of the axial bundle of the optic nerve and along the central vessels into the disk. Paton and Gordon Holmes believe that their observations establish the fact that papilledema is an edema of the nerve-head due to two factors—venous engorgement and lymph stasis. Carl Behr maintains that choked disk follows passive lymph stasis brought about by virtue of a compression of the optic nerve because of an interruption of its lymph passages which are proceeding centrally, and that this takes place, for instance, if the intracranial pressure is elevated by a tumor.

In general terms it is probable that choked disk is produced by a combination of factors. In this combination increased intracranial tension or pressure is by far most prominent, and the mechanical theory of its pathogenesis affords the most satisfactory explanation. If other factors are potent, they have not yet been definitely discovered.

Inflammatory or irritative processes in the optic nerve and its sheaths sometimes have an active influence, and if the inflammatory condition predominates, the elevation of the disk is less marked and the process is apt to extend to the retina, where exudations and hemorrhages are visible; in other words, the lesions warrant the descriptive term *inflammatory optic neuritis.*

Pathologic Anatomy.—In the early stage of true choked disk the edema is non-inflammatory and no evidences of inflammation are present. In later stages moderate inflammatory infiltration is evident. There may be blood extravasations, swellings and varicosities of the nerve-fibers, and slight cellular exudation along the thickened and dilated vessels. In the *interstitial* form of *neuritis* the inflammation begins in the sheath and septa, with the formation, in addition to the edema, of an exudation rich in cells, which subsequently organizes. There follow thickening of the interfascicular septa, increase of the nuclei, and degeneration and atrophy of the nerve-fibers from pressure. In some cases degeneration of the ganglion cells of the retina is evident, depending upon the fact that an arterial branch supplying that particular area has been occluded. Such degenerative areas may give rise to scotomas or sector-like defects in the visual field.

An ampulliform dilatation of the optic-nerve sheath posterior to the eyeball is found in a certain number of cases, and in addition to distention of the intervaginal space there may be an infiltration of small cells in the sheath.

1. **Optic-nerve Atrophy.**—Under the general term *atrophy of the optic nerve* are included the various types of degeneration and shrinking of the fibers of the optic nerve, usually described under the subdivisions *primary, secondary, consecutive* (*neuritic* or *postpapillitic*), and *retinal* and *choroiditic atrophy*. The last are really forms of consecutive atrophy.

Symptoms.—Certain general symptoms are common to optic-nerve atrophy, although these are subject to variations according to the clinical types.

1. *Changes in the Nerve-head.*—(*a*) *Alterations of the Normal Color of the Disk.*—The color of the disk varies from a slight gray pallor to a pure gray, greenish-gray, or entirely white ("paper white") hue. There may be a commingling of gray and red, producing the so-called "grayish-red disk," and often there is a decided greenish tinge, rarely a blue one.

Grayness of the optic nerve is not always detected by ordinary methods of examination, especially in the deeper layers of the disk, but if the fundus is examined by means of properly regulated illumination, and through a lens which neutralizes any error of refraction, this deep pallor is usually evident. It is important to employ both the direct and indirect methods of examination, and the concave and plane ophthalmoscopic mirror. Ophthalmoscopy with the diaphragm lamp has been described (page 113).

(*b*) *Alteration in the Center of the Disk.*—Sinking of the surface of the disk, varying from a slight depression to a complete excavation (see page 414), occurs according to the degree of degeneration which the nerve-fibers have experienced. The shape of the excavation depends somewhat upon the presence or absence of a normal physiologic cup. At the bottom of the atrophic excavation the mottling of the lamina cribrosa is distinct in some cases of atrophy; in others it is not apparent, the center of the disk may be filled in.

(*c*) *Alterations of the Margins of the Disk and of the Scleral Ring.*—In complete atrophy the margin of the optic disk is unusually distinct. In the atrophy which follows a neuritis or retinitis, however, the margins are often veiled for a long time.

Undue broadening of the scleral ring indicates shrinking of the disk. Even in the early stages of spinal atrophies the disk may be surrounded by a broad scleral ring, which, taken into consideration with alteration in the color of the papilla and contraction of the color-field (especially red and green), affords diagnostic aid in the study of gray degeneration of the optic nerve.

2. *Changes in the Vessels.*—In simple atrophy, while there may be narrowing of the vessels, this is not always the case, and certainly not in the manner seen in consecutive atrophies. Sometimes the arteries are narrowed and the veins unchanged.

In neuritic (consecutive) atrophy the arteries are much contracted

and the veins in contrast are larger than usual, often retaining some of the tortuosity which was so marked a feature during the papillitic stage. By the contraction of the tissue these, too, may later become narrowed. Development of white tissue along the course of the vessels, due to thickening of the perivascular lymph-sheath, is common in this form of atrophy.

In retinitic and choroiditic atrophy there is marked contraction of both veins and arteries, which are also diminished in number.

3. *Changes in the Surrounding Eye-ground.*—The presence of alterations in the general fundus depends entirely upon the cause of the atrophy. In simple gray and white atrophy such signs may be absent; but in postpapillitic and retinitic atrophy, spots of degeneration, marking the places of former hemorrhages and exudations, and patches of pigment heaping, are commonly seen.

In addition to these ophthalmoscopic changes the following symptoms occur:

1. *Change in Central Vision.*—This varies from a slight depreciation to blindness, and, if the atrophy is bilateral, is usually more marked in one eye than in the other. In every case, where this is possible, especially in early cases or cases of doubtful atrophy, any existing refractive error should be corrected before deciding the degree of depreciation of central sight.

2. *Change in Light-sense.*—Usually it has been found in pure optic-nerve atrophy that the light-difference is defective, but the light-minimum not much influenced. *Adaptation*, or the accommodation of the eye to varying degrees of illumination, is greatly restricted, for example, in tabetic optic-nerve atrophy (Lohmann).

3. *Change in the Field of Vision for White.*—The following changes occur: Concentric contraction; very irregular limitations presenting large re-entering angles (peripheral scotomas); quadrant-shaped defects; complete loss of one-half of the visual field (hemianopsia); and an abnormal blind-spot in the center of the field (central scotoma) or adjacent to it (paracentral scotoma).

The field of vision, concentric restriction being most common, does not give evidence of the cause of the atrophy, although it may afford information of the localization of the defect; thus, an affection of the macular fibers will produce a central scotoma. In spinal atrophy the limitation more frequently begins at the outer side than in other situations.

4. *Change in the Field of Vision for Colors.*—There is always a defect in color vision. Usually there is, first, contraction of the green field, then of the red, and afterward of the blue and the yellow fields. In late stages of optic-nerve atrophy color-sense is abolished. Occasionally the perception of red becomes defective before that of green is influenced by the atrophic process.

Generally the contraction of the color field is much greater than that of the white field (compare page 434). Central vision may be good, the white field but slightly or not at all affected, and yet the

green and the red fields may be considerably contracted. Hence the importance of combining all these examinations before deciding whether decoloration of the papilla is pathologic or not.

4. *Changes in the Pupil.*—The relations of the pupil to the action of light depend upon the degree of atrophy. In many cases there is more or less perfect paralytic mydriasis, and when the atrophy is complete the pupil is dilated and the iris motionless. Even when the pupil fails to contract under the influence of light thrown upon the retina, it may do so in the act of convergence. (See also page 65.)

If the atrophy is confined to one eye, no reaction occurs when the light falls upon the corresponding retina, but instant contraction takes place when this is directed upon the retina of the opposite (unaffected) side. The pupil changes in spinal disease (tabetic atrophy) have been described (see page 66).

Varieties of Optic-nerve Atrophy.—1. *Primary Atrophy (Sometimes Called Gray, Progressive, Spinal or Tabetic Atrophy).*—The color of the disk is gray or white; sometimes it has a greenish or bluish tint; the decoloration is associated with translucency, and the stippling of the lamina is evident; the excavation, if it exists, is complete and saucer-like; the vessels either are smaller than normal, especially the arteries, or they are unaffected in size; the edge of the disk is sharply marked, and the scleral ring clean cut all around. These symptoms describe the fully formed atrophy.

In the earlier stages of the degeneration, according to the late Dr. W. F. Norris, the disks are of a dull red tint, their capillarity is superficial, and the deeper layers, in the neighborhood of the lamina cribrosa, are gray and wanting in circulation. There is often sufficient haze of the retinal fibers to veil the scleral ring. Later the nerves become pallid, are somewhat woolly superficially, and are surrounded on all sides by broad and sharply cut scleral rings. The larger retinal arteries and veins do not at this stage present any appreciable change in their caliber or appearance. Both eyes usually are affected, one showing a further advance of the degenerative process than its fellow.

2. *Secondary Atrophy.*—The color of the disk may be gray and assumes a tint not greatly dissimilar from the atrophy which has just been described. In other instances the color is more decidedly white. Both sets of vessels may be contracted, usually the veins being less affected than the arteries. In a certain number of cases of secondary atrophy it is probable that preceding the degenerative stage there is a transient congestion of the disks; certainly this is true in those cases where there has been a retrobulbar neuritis.

3. *Consecutive Atrophy.*—(*a*) *Postpapillitic Atrophy.*—The color of the disk is very gray or white, sometimes with a decidedly greenish tinge or even a blue tint. It is noticeable, however, that the translucency present in the primary form of atrophy is absent, and the stippling of the lamina cribrosa is not visible, owing to the existence of a non-transparent tissue which covers it. The borders of the disk are slightly veiled, and the perivascular lymph-sheaths are thickened. The arteries

are contracted, the veins frequently exhibiting distinct tortuosity. Retinochoroidal changes are often evident (see also page 547, paragraph 5).

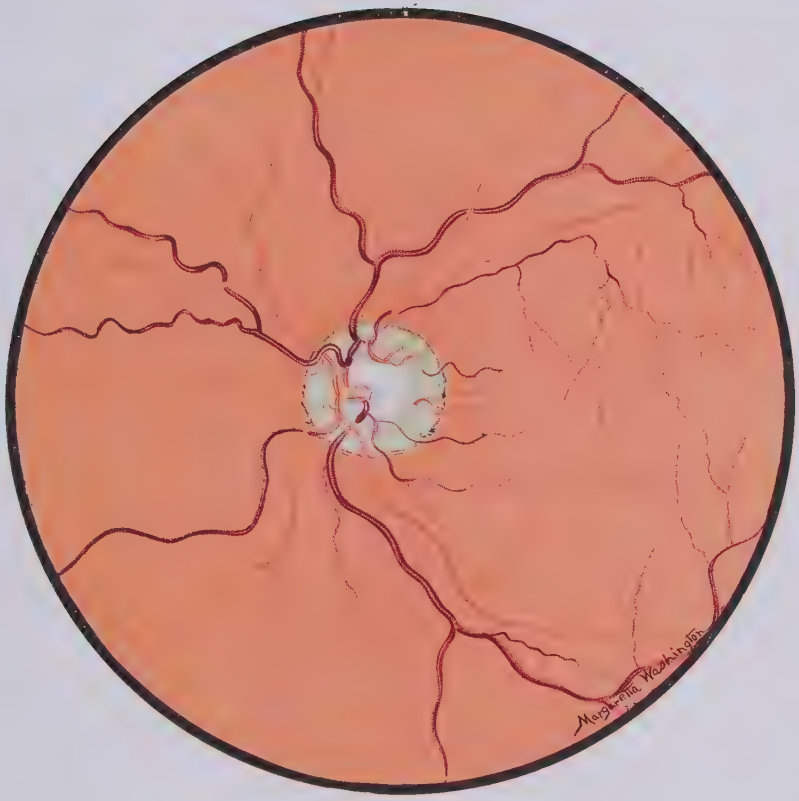
(*b*) *Retinitic and Choroiditic Atrophy.*—This is in the form of atrophy of the nerve to which reference has already been made, and which follows severe forms of retinitis and choroiditis. The disk has a distinctly yellowish tinge, being somewhat waxy in appearance; its borders are not sharply marked, and the vessels are narrowed, often to a great degree.

Causes.—In addition to the forms of atrophy which follow papillitis and papilledema (*postpapillitic atrophy*), inflammation of the choroid and retina (*choroiditic* and *retinitic atrophy*), embolism and thrombosis of the central artery and thrombosis of the central vein of the retina (*embolic* and *thrombotic atrophy*), the varieties which are gathered under the general terms *primary* and *secondary* atrophy require further mention.

Primary atrophy of the optic nerve occurs, in the great majority of instances, in diseases of the spinal cord, and especially in locomotor ataxia. It is frequent in general paralysis of the insane and insular sclerosis, but less common in lateral sclerosis. There is some difference of opinion in regard to the frequency of optic-nerve atrophy in locomotor ataxia, but an average of a number of observations gives 33.7 per cent. of atrophies. In most instances it begins in the preataxic stage. To one variety of the affection, in which the atrophic process precedes, often by long intervals, the ataxic symptoms, the name *optic-nerve type of tabes dorsalis* is often applied. The visual fields of tabetic atrophy of the optic nerve have been especially studied by Uhthoff, Stargardt, and Leslie Paton. They present a variety of defects, for example, peripheral loss of white and colors, peripheral loss of colors with full fields for white, loss of sectors, central scotoma (but difficult to eliminate the influence of tobacco), and hemianopsia (probably not in uncomplicated cases, although Fuchs maintains it may be an essential tabetic process). Paton believes the variability of the fields depends upon variability of the pathologic process, that is, whether the attack of the toxin is more severe on the parenchymatous or on the interstitial tissue of the optic nerve. Central scotomas are commonly observed in disseminated sclerosis. Optic-nerve atrophy may also be associated with Friedreich's ataxia, amyotrophic lateral sclerosis, chronic myelitis, paralysis agitans, spastic spinal palsy, and bulbar palsy.

Primary atrophy has also been ascribed to the influence of cold, imperfect nutrition, disturbed menstruation, and venereal excesses. It may be caused by diabetes, syphilis, pellagra, the toxic action of certain drugs, antityphoid vaccination (Terrien), and excessive hemorrhage (see also page 578). Its association with deformities of the skull has been described (see page 550). Undoubtedly it may be due to arteriosclerosis, the thickening of the arterial wall closing the lumen of the vessels. Very rarely it has resulted from sclerosis of the central artery of the retina. Optic-nerve atrophy in old people without discoverable constitutional

PLATE VII.



Primary atrophy of the optic nerve.

or local cause for its existence other than arteriosclerosis is not very uncommon, but not usually far advanced. Such nerve changes may be due to sclerosis of the nutrient vessels of the optic nerve.

Secondary atrophy appears under the influence of compression of the optic tract and the optic fibers—for instance, by internal hydrocephalus or by pressure of a tumor, pituitary neoplasm or struma, exostosis, or aneurysm upon the chiasm. It is also said to occur with meningitis without preceding neuritis. Optic-nerve atrophy in patients who give a history of cerebral symptoms in early life is not infrequently encountered and is probably the result of a chronic meningitis from which recovery has taken place; such atrophies doubtless in most instances are the result of preceding neuritis, and in this sense are consecutive and not secondary atrophies. Compression around the optic foramen is likely to produce secondary atrophy by direct injury to the fibers of the optic nerve. Blows on the head, especially in the neighborhood of the supra-orbital foramen, causing fracture of the orbital plate or periostitis, may be followed by a like result. Atrophy may result from inflammation of the axis of the nerve posterior to the eyeball.

Pathologic Anatomy.—The nerve-fibers lose their medullary sheaths and are converted into fine fibrillæ, between which are numerous fatty granular cells; no true inflammatory process appears. Later all nervous elements may disappear. Tabetic atrophy of the optic nerve has been ascribed to disease and disappearance of the retinal ganglion cells, but from a comparison of the visual fields in glaucoma with those in tabetic atrophy Rönne concludes that the lesion is in the optic nerve-fibers, and Stargardt seems to have demonstrated than an exudative process in the chiasm and nerves precedes the degenerative one. In postneuritic atrophy there is considerable new-formed connective tissue in the nerve-head and trunk through which run the thickened vessels; the sheaths of the nerve-fibers degenerate, break down into fine drops, and the nerve-fibers become varicose and either shrink or disappear. The septa are much thickened, and in advanced cases the nerve becomes a narrow, purely connective-tissue cord.

Diagnosis.—The student is warned not to mistake the pallor of age for the pallor of disease; not to mistake a large physiologic cup, with its margin shelving toward the temporal border of the disk, for an atrophy confined to half of the optic papilla; and not to mistake small patches of retained marrow-sheath for atrophic changes.

Not every gray disk, with an unusually marked scleral ring, is indicative of atrophy, and it is only when these appearances accord with the other manifestations of beginning degeneration that the diagnosis of incipient atrophy is justified. The Wassermann test of the blood and of the spinal fluid should always be employed, and a stereoscopic *x*-ray plate should be made in order to determine the condition of the pituitary region and the accessory sinuses of the skull; in short, a thorough examination in all respects should be made in each case of optic-nerve atrophy.

The differential points between a chronic glaucoma and an optic-nerve atrophy have been described (see page 434), and also the relation of light-sense to optic-nerve atrophy. According to Behr, in tabetic optic-nerve atrophy there is always a decided disturbance of *dark adaptation*, which may be evident prior to change in the visual field or the color sense.

Course and Prognosis.—The course of optic-nerve atrophy is usually a slow one, lasting for months and it may be years, depending to a certain extent upon the original cause of the atrophy. Exceptions to this statement concern those forms of atrophy which follow injury (fracture at the base of the skull or at the apex of the orbit), where the process may rather quickly develop. Even in these cases a week or more may elapse before ophthalmoscopic atrophy is visible.

The prognosis is unfavorable in primary or, as it is sometimes called, progressive atrophy, the tendency being to a gradual deterioration of sight with shrinkage of the field of vision, until blindness is the result. The prognosis of a consecutive atrophy depends entirely upon the amount of damage which is likely to ensue from the shrinking which follows during the subsidence of the neuritis. In the forms of atrophy which follow an inflammation of the axis of the nerve the prognosis is better.

Treatment.—This depends upon the cause. If syphilis is present, the usual remedies are indicated; but mercury is useless in advanced cases, even in syphilitics. While salvarsan or neosalvarsan exercise no detrimental effect on a healthy optic nerve, at one time it was maintained that salvarsan exerted an evil influence on tabetic atrophy. Recently this contention has been set aside and there are now a number of observations on record which tend to show that neosalvarsan or, its equivalent, neo-arsphenamin, if used early and while color perception is still good, represents a therapeutic agent of value in the treatment of tabetic atrophy. *Salvarsanized serum* injected intraspinously (Swift-Ellis method) has been followed by improvement in tabes, but the response of optic-nerve atrophy to this form of medication has not been as satisfactory as it originally was hoped it would be.

Intracranial, that is, *intracisternal injections* of bichlorid of mercury appear to be more efficacious than intraspinous injections of salvarsanized serum. Recently, they have been advocated by Sanford R. Gifford ($\frac{1}{50}$ grain of bichlorid of mercury being introduced into this lateral ventricle and repeated as required), who prefers this method and who reports, as the result of it, improvement in vision. The best results were obtained in early cases and where there was little evidence of other nervous involvement.[1] It is vitally important that syphilitics from the earliest manifestation of their infection should be systematically and repeatedly examined from the ophthalmic standpoint.

[1] Consult Diagnosis and Treatment of Luetic Involvement of the Optic Pathways by M. J. Schoenberg, Trans. Amer. Ophth. Soc., xvii, 1919, and Intracisternal Injections in the Treatment of Luetic Optic Atrophy, by Sanford R. Gifford, Brit. Jour. Ophth., 1923, vii, 506.

Strychnin has been much employed administered in full doses, preferably by the hypodermic method; it may be enforced by nitroglycerin. Other remedies, according to the cause, are iodid of potassium, nitrate of silver, phosphorus, arsenic, iron, santonin, lactate of zinc, hypodermics of antipyrin (Valude), and injections of organic liquids, all of doubtful value. Negative galvanism has been advised, and good results have been reported by L. W. Fox, Ziegler, Radcliffe, F. W. Coleman, and many other observers. Coleman especially recommended the *sinusoidal current*, a binocular electrode being placed over the eyes and an oval pad to the nape of the neck. The treatment should take place for twenty minutes each day. High-frequency currents have been advocated and they should be tried. In a few instances suspension is said to have been followed by improvement of vision in tabetic atrophy. There is no satisfactory evidence that radium and the Roentgen rays are useful therapeutic agents in the treatment of optic-nerve atrophy.

Hereditary Optic-nerve Atrophy (*Hereditary Optic Neuritis*).—A remarkable type of optic-nerve atrophy, that is, of the papillomacular bundle (retrobulbar neuritis), first systematically described by Leber (*Leber's disease*), is hereditary, and may appear for a number of generations usually, but not always, in the male members of the family. It is transmitted by unaffected females. An affected male seldom transmits the disease (Nettleship). The disease generally begins between the eighteenth and twenty-third year, but has been observed as early as the fifth year and delayed as late as the sixty-seventh year. According to William F. Norris, there are three stages of the affection: (1) Stage of edema and congestion of the disk; (2) stage of gray discoloration of the nerve-head, and (3) stage of pronounced atrophy. The condition is usually symmetric, both eyes being affected at the same time, or there may be an interval of some weeks. Central scotomas are commonly present and are usually permanent. In Arnold Knapp's cases the first generation presented central scotomas and peripheric contractions of the visual fields; but in the second and third generations no central scotomas developed. The subjects of this disease may have headaches, tremors, vertigo, and epileptic attacks. The incidence of the affection may be at the time of sexual development or at the time of sexual decay. Disturbances in the region of the hypophysis (*x*-ray examination), first noted by W. S. Bruner, in their relation to Leber's disease have attracted attention, and T. Herbert Fisher suggests that a disorder of the pituitary body of temporary duration and moderate degree may be adequate to explain the phenomena of this affection, rather than a family tendency on the part of the papillomacular bundle to degeneration or to inflammatory attack. Enlargement of the sella turcica has been noted by a number of observers (Fisher, James Taylor, Pollock, E. T. Collins, Zentmayer, and Pancoast); sometimes, however, it is absent. Collins classifies the disease among the *abiotrophies*. Usually treatment has little or no effect, but improvement and even recovery have been reported (Cargill). Thyroid and pituitary body extract should be exhibited.

Orbital Optic Neuritis (*Retrobulbar or Axial Neuritis; Central Amblyopia*).—In contradistinction to the optic neuritis which is specially localized at the intra-ocular end of the nerve, an inflammation occurs in the orbital part of the optic nerve, which is called *orbital optic neuritis—retrobulbar* or *axial neuritis*. It appears in an *acute* and a *chronic* type.

1. *Acute Retrobulbar or Axial Neuritis.*—The symptoms of this affection are the following: Obscuration of vision, beginning in the center of the visual field, and sometimes rapidly progressing in from one to eight days to complete or nearly complete blindness; at first negative ophthalmoscopic appearances, later blurring of the margins of the disk, hyperemia of its surface, and sometimes, in severe cases, diminished caliber of the retinal arteries and fulness and pulsation of the retinal veins; distinct pain on movement of the eyeball, or when the globe is pressed backward into the orbit.

The scotoma does not always either expand to the limits of the visual field or remain in its central position with exactness. Rönne has described a "shifting of the visual field defect" at different periods in the course of the disease. In many cases, although the process is acute, in the sense that the visual defect develops promptly, the area of the field involved does not exceed the boundaries of the 10- or, at most, the 20-degree circle of the chart.

The affection depends upon a neuritis, most severe in the optical canal, and at first chiefly located in the papillomacular tract, from which it may extend, however, until the whole diameter of the nerve is involved. If the process is unchecked, necessarily secondary degeneration of the nerve-fibers takes place. There is also degeneration of the ganglion cells of the macula.

Cause.—The determining cause of the disease is the presence in the blood of an infecting agent existing in association with some disease—for example, rheumatism, dysentery, intestinal sepsis, tuberculosis, gout, syphilis, influenza, diabetes, smallpox, and scarlet fever; or coming directly from a focus of infection in the mucous membrane of the nose, the mouth (the teeth), the tonsils, the ethmoid cells, the sphenoid sinus, and other sources of focal sepsis; or arising as the direct result of an inflammatory process in the orbit—*e. g.*, cellulitis, or in the optic canal—for example, periostitis, gummatous deposits, etc. The disease has also been attributed to certain toxic agents, such as alcohol, lead, etc.; to menstrual disturbances, especially sudden suppression of the menses, to lactation and pregnancy, to auto-intoxication, and to overwork and prolonged eye-strain. Not only may retrobulbar neuritis be caused by suppurative ethmoiditis, but it may arise in connection with hyperplasia of the ethmoid bone (Vail). A certain number of cases exist for which no cause can be ascertained. Nettleship divided cases of retrobulbar neuritis into two groups: the *idiopathic*, in which the disease starts in the nerve itself, and *symptomatic*, in which it is communicated to the nerve by the surrounding tissues.

This disease may be part of the symptomatology of multiple scle-

rosis and of acute or subacute myelitis, and is in these circumstances of most serious prognostic import. Retrobulbar neuritis ipsolateral to the lesion has been recorded as a symptom of tumor or abscess of the frontal lobe (Paton, F. Kennedy); choked disk may be present in opposite eye. The author has observed several cases of this character.

The *course* of the disease, as previously noted, is not always rapid or fulminant, as it is called, for example, in insular sclerosis, where the central field defect is limited in extent and the activities of the acute expansive scotomas do not appear. It is sometimes bilateral, but more frequently unilateral, or a long interval may occur between the involvement of the first and second eye. Relapses may occur, and the affection may alternate between the two eyes. As was pointed out by Mr. Marcus Gunn, there is marked analogy between axial inflammation of the nerves in the optic canal and paralysis of the facial nerve (Bell's palsy) when its trunk is involved in its tortuous course through the wall of the skull. Indeed, as the author has shown, retrobulbar inflammation may be pre-

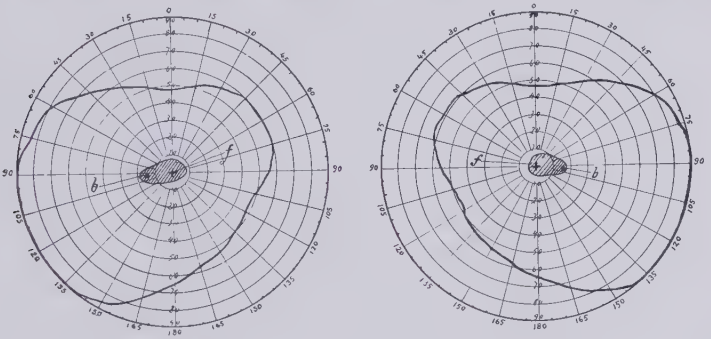


FIG. 251.—Papillomacular scotomas from a case of tobacco amblyopia: *f*, Fixation; *b*, blind-spot.

ceded by an attack of peripheral facial palsy, either upon the same or the opposite side.

Prognosis.—Although this must always be guarded, in the majority of instances the tendency is to recovery, and, under careful treatment, to perfect recovery. In severe cases permanent pallor of all or part of the optic nerve, defective central vision for colors, central scotoma, and contraction of the peripheral field may remain. The fact that retrobulbar neuritis may indicate the future onset of *disseminated sclerosis* should not be forgotten. It may precede the other symptoms by many years. According to Marx, it may develop in a certain percentage of the cases from one to seven years after apparent recovery. Shumway's study of retrobulbar neuritis dependent on focal infection followed by insular sclerosis is most suggestive and important. Stark has made similar observations. There is also a variety of the disease due to exposure, menstrual disturbances, and rheumatism, in which the same symptoms appear as those previously described, but all of a milder type and all more amenable to treatment.

Treatment.—In so far as possible the cause must be eliminated. If the affection has occurred during the course of an acute infectious disease, the treatment of this particular malady is indicated. In other circumstances the best results follow active diaphoresis, full doses of salicylic acid, the free use of mercury, the iodids, and counterirritation on the temple. No patient with retrobulbar neuritis should escape searching examination of the nasopharynx, the ethmoid, frontal and sphenoid sinuses. If purulent disease is found, operation with suitable drainage may speedily relieve the ocular condition; if this is neglected blindness may result. (See also page 672.) Retrobulbar neuritis of tuberculous origin has been relieved by injections of tuberculin (Igersheimer). The affection may be due to syphilis and therefore the proper serologic tests should always be made and treatment directed according to the result.

2. *Chronic Retrobulbar or Axial Neuritis (Tobacco Amblyopia; Toxic Amblyopia).*—The clinical symptoms of this affection are as follows: Diminution of sight, associated with fogginess in the center of the field of vision, unimproved by glasses and most noticeable in bright light; reduced acuteness of vision, which varies from $\frac{6}{9}$ to counting fingers; negative ophthalmoscopic appearances or pallor of the temporal half or of a quadrant-shaped portion of the papilla; normal peripheral boundaries of the field of vision; symmetric color scotomas (relative or absolute, according to the stage and severity of the process), especially for red and green, but also for blue, usually oval in shape, stretching from the fixing-point to the blind-spot, and rarely passing much to the nasal side of the former; defective light-sense, the light-difference (L. D.) being particularly affected. The scotoma, which is the most important of the symptoms, represents a reddish-green blind area, and commonly the extent of green-blindness is greater than that of red, which, in its turn, may be surrounded by an area of imperfect color-sense. Sometimes its beginning is a small, easily overlooked scotoma exactly over the fixing-point (Groenouw). After the typical egg-shaped scotoma is developed, the process may cease, or there may be a stage of progression characterized by an increase in the size of the color defect, usually above, until it meets the limit of the red field; that is, the scotoma has "broken through." Absolute defects may be found, or the entire scotoma may become absolute. The periphery of the visual field is not always intact, and contractions may be found if the tests are made under diminished illumination. According to Dorrell and J. Herbert Fisher the sensory reflex is much diminished or entirely absent in toxic amblyopia.

Recent investigations, modern methods of scotometry being used, indicate that our conceptions of the form and character of the scotoma in tobacco amblyopia, as described above, must be in some particulars altered. Thus P. G. Doyue has developed three types: a large scotoma which involves the blind-spot and the area between it and fixation, stopping just short of the actual fixation point; a scotoma close to fixation point, connected by a relative area to the blind-spot which may or may not extend toward fixation; and a scotomatous finger pointing from the

blind-spot toward fixation. In general terms he found that blue conformed with white and red with green; except in slight cases the area in which the perception of red is imperfect involves the fixation point. C. H. Sattler, investigating 26 cases of tobacco amblyopia, found that the spot of the greatest density of scotoma did not touch the fixation point in half of them. He would abandon the term "central scotoma" and for it substitute the term *papillomacular scotoma,* originally suggested by Sachs.

Causes.—The most important drugs and toxic substances which may be responsible for the clinical symptoms of so-called intoxication amblyopia are tobacco and alcohol, either singly or combined, stramonium, cannabis indica, thyroid extract, chloroform, chloral, opium, bisulphid of carbon, nitrobenzol, carbon monoxid and other poisonous gases, arsenic, atoxyl, lead, and iodoform. Of the substances mentioned, tobacco is the one most often responsible for this affection, but as the users of tobacco are also usually consumers of alcohol it is difficult

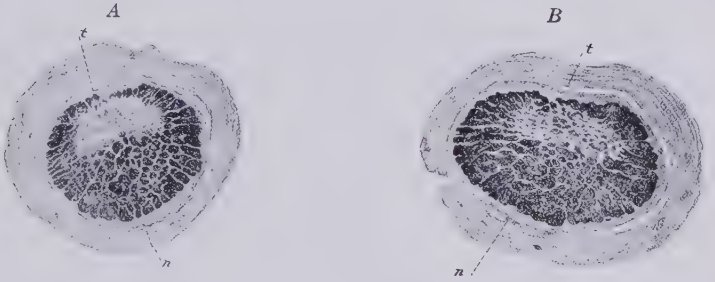


Fig. 252.—Sections of the right optic nerve in a case of toxic (tobacco) amblyopia showing degeneration of the papillomacular bundle (Weigert's stain): *A*, Transverse section of the optic nerve 13 mm. behind the globe; *B*, transverse section of the optic nerve in the region of the optic foramen.

to separate the etiologic influence of these two drugs, and hence the name *intoxication* or *toxic amblyopia* is used to describe a central amblyopia which may be due to either of these substances or to their combined influence. A *pure* tobacco amblyopia, which the author believes he has observed, is uncommon; indeed, some observers deny its existence. So, too, a *pure ethyl alcohol amblyopia,* due to the abuse of "spirits," is encountered; multiple alcoholic neuritis may be a complication. Although almost always bilateral, a few instances have been recorded in which a symmetric development of tobacco amblyopia has not been evident, but it is doubtful if a purely unilateral case has been observed. It is rare before the thirty-fifth year. The features of the scotoma of tobacco amblyopia are distinctive, in that it is a typical centrocecal defect with sloping margins which is never really central (pericentral) in situation and which contains within it one or two areas of greater intensity, the defect for red being far greater than that for white. These characteristics serve to distinguish it from other "toxic

scotomas" in the central area of the field. The studies of Traquair in this respect are especially satisfying.

Chronic axial neuritis is also caused by toxic agents elaborated in the cause of general diseases, for instance, diabetes (page 577) or intestinal sepsis. The subjects of pellagra may have pallid disks and central relative scotomas (Calhoun). Even in these circumstances it is not always possible to eliminate the influence of tobacco, and if a proper analysis of the scotoma should reveal features already described, it would seem that tobacco should be accused as the responsible factor. Also in syphilis, sinus disease, and other infections, although the signs of acute neuritis may be absent, the interpretation of the axial degeneration may be a relative scotoma such as has been described. Although the relation of alcohol and tobacco to the type of amblyopia which has been described is apparently well established there is little or no doubt that disturbances of metabolism, gastric and intestinal sepsis, and a toxemia derived from areas of focal infection are large contributing factors, in that they lessen the resisting power of the papillomacular bundle to the influence of the two agents named. This was long ago pointed out, at least in so far as intestinal and gastric troubles are concerned, by Sachs, and it should receive more attention than is usually given.

The pathologic lesion which causes this form of amblyopia, according to Uhthoff and other observers, is an interstitial inflammation of the *papillomacular* fibers of the optic nerve. These fibers, traced by means of their degeneration, consist of a bundle shaped like a triangle near the eye, with its base in the lower and outer part of the nerve, and its apex at the central vessels. Gradually it passes to the center of the nerve, which it reaches in the optic canal. Finally, it can be followed into the chiasm and tracts. Nuel and others believe that the so-called central toxic scotoma is not caused primarily by a neuritis of the macular bundle, but represents a disease of the macula lutea, causing degeneration of its cells, and that the optic nerve changes are secondary to destruction of the nerve-cells in the macula. The investigations of Birch-Hirschfeld led him to doubt that the process depends upon a primary interstitial inflammation of the optic nerve. He believes that there is a primary involvement of the nervous elements of the nerve and retina, with an accompanying proliferation of the glia and increase in the connective tissue. Schieck concludes that the disease begins with an alteration of the blood-vessels within the optic nerve, and as the central fibers are less liberally supplied with blood, they are the first to be affected. While vessel disease may have an important bearing on this affection, a direct action of the toxin on the nervous elements seems undoubted, and toxic amblyopia may be regarded in all probability as a ganglion-cell affection rather than one primarily of the nerve-fibers.

Course and Prognosis.—The course is, as its name indicates, a chronic one, but the prognosis of the tobacco and alcoholic cases is good, provided the patients present themselves at an early enough stage for treatment. In rare instances complete optic-nerve atrophy results.

Treatment.—This consists in total abstinence from the use of tobacco and alcohol, and in the earlier stages this alone will be sufficient to bring about a cure. Later, a remedy of value is strychnin, which, as in other instances of optic-nerve disease, should be pushed to its full physiologic limit. In order to help in the absorption of inflammatory products, iodid of potassium may be given. Regulation of diet and free diaphoresis are valuable measures. Sinusoidal galvanism applied over the eye with a properly constructed electrode appears to act favorably. Examination of the urine, as the author and David Edsall have shown, is apt to reveal an excessive excretion of enterogenous decomposition products, and with its restoration to normal, under the influence of proper dietetic regimen, the eye conditions improve. The patients should drink water freely. Temporary improvement occurs under the influence of inhalations of nitrite of amyl, and the circulation of the optic nerve may be stimulated by the exhibition of digitalis and nux vomica. Lecithin has been recommended in the treatment of tobacco amblyopia (H. de Waele).

Necessarily, if some poison other than alcohol or tobacco is active, the patient must also be removed from its influence.

Senile Changes in the Optic Nerve (*Senile Amblyopia*).—The senile vascular alterations in the retina have been described (page 521). We are indebted to E. Fuchs for a study of changes in the optic nerves of old persons. Frequently amyloid bodies develop, just as they do in the central nervous system, from the neuroglia. They may displace the nerve bundles and damage them. Pressure of a sclerotic internal carotid or ophthalmic artery may injure the nerve; atrophic foci are often found, probably due to arteriosclerosis (see also page 519). The disk may be quite pallid, but not actually atrophic, and the vessels diminished in size. A central scotoma may occur suddenly in elderly persons whose arteries are diseased (Traquair).

Injury of the Optic Nerve.—This may be produced by the entrance of a foreign body into the orbit, for example, a splinter of wood, a bullet, etc., or by a fracture involving the bony wall of the orbit or base of the skull, or by the thrust of a knife. Atrophy of the optic nerve is the result. Indirect injury of the optic nerve may be caused, as Evans has shown, by a blow in the region of the external angular process of the frontal bone. The primary impairment of vision and loss of the temporal field may be followed by atrophy of the nerve.

During the past war there were many optic nerve injuries as the result of the passage of a missile through the posterior part of the orbit causing incurable blindness. *Avulsion of the optic nerve* was not infrequent. After the hemorrhage following such an accident absorbs a traumatic excavation is visible—a kind of "coloboma or surgical conus," as Lagrange calls it. This may be filled up later with proliferated connective tissue. The retinal vessels, although they may disappear, may in some cases remain. This has been often noted, and has been recently well figured and commented upon by Edward Jackson. Parsons states the vessels refill through direct and indirect cilioretinal

anastomosis, the blood being derived from intact anterior and posterior arteries. It is not necessary that there shall be a division of the nerve

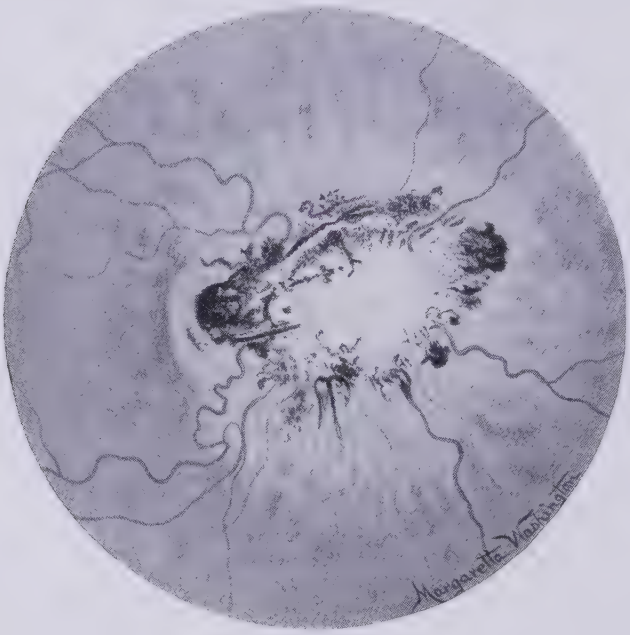


Fig. 253.—Avulsion of the optic nerve (from a patient in the University Hospital).

sheath as a factor in the production of avulsion (W. T. Lister and M. L. Hine).

Tumors of the Optic Nerve.—These usually are divided into *intradural* and *extradural* tumors. Of the former, 102 cases have been collected by W. G. M. Byers in his notable monograph on this subject, and they include fibroma, sarcoma, glioma, endothelioma, gumma, tubercle, and myxoma. Of the latter (extradural tumors), Parsons has been able to find 12 undoubted cases, and of these, 9 were almost certainly endotheliomas. Hudson in 1912 analyzed 154 cases collected from the literature, and classifies 118 of them as gliomas, or as probably gliomatous, 6 as fibromatosis, and 29 as probably endotheliomas. Intracranial extension may take place; this was found after death in 15 cases in Hudson's list. Hudson and Verhoeff object to the terms "intradural" and "extradural" tumors. Verhoeff's classification is: tumors arising in the nerve stem (intradural), and tumors arising in the nerve sheath (extradural). According to him, the most common tumors of the optic nerve are gliomas.

Symptoms.—Exophthalmos is a prominent feature, the eye being pushed downward and forward; the motion of the globe is unaffected, and defective vision is an early manifestation. The growth is slow and painless, but sometimes a suppurative keratitis may result. The ophthalmoscope reveals distended veins, retinal edema, and papilledema

followed later by atrophy and shrinking of the vessels. Atrophy of the papillomacular bundle and central scotoma may occur, as in one of the author's cases. Increasing hyperopia is a sign of importance;

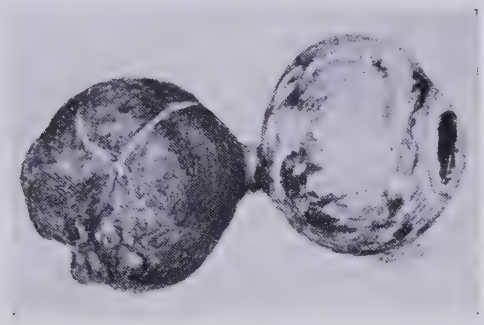


Fig. 254.—Psammosarcoma of the optic nerve.

it is, however, not confined to tumors of the optic nerve, as it may be observed as a symptom of orbital growths (Stieren). With intradural tumors the movement of the eye is usually restricted in the opposite



Fig. 255.—Microscopic section of a nerve-head containing hyaline bodies (from a photomicrograph).

direction to any modification of the proptosis directly forward (Parsons).

Treatment.—In many instances the eyeball must be removed with the tumor, but occasionally the globe can be saved (11 times in Finlay's

collection—subsequent loss in 4 cases). Exenteration of the orbit has been necessary, primarily, in a few instances, and secondarily on account of local recurrence. Even after enucleation, and sometimes at a period long removed from the time of operation, the growth has recurred and death may occur from intracranial involvement. Since the introduction of Krönlein's operation and its modifications exploration of the orbit has been greatly facilitated and the opportunity of saving the eyeball has been increased. A. Knapp and R. Reese report satisfactory results after a Krönlein operation. Hudson records the reports of 43 operations (Krönlein's method or without bone resection), with 15 first class results; complications described are ptosis, deviation of the eye, phthisis bulbi, and meningitis.

Tumor may develop in the prechiasmal segment of the optic nerve in the neighborhood of the optic foramen and may be the cause of blindness (W. E. Dandy).

Hyaline Bodies in the Papilla.—This affection is characterized by the formation in the optic papilla of small excrescences or globular formations, which are sometimes described as colloid masses. The bodies are variously shaped, chiefly roundish, of a yellowish-white or bluish-gray color, forming a mulberry-like appearance and presenting a striking ophthalmoscopic picture. They may occur at any age of life, sometimes in association with choroidoretinitis, optic neuritis, and optic-nerve atrophy, but also in eyes free from other pathologic changes and with perfectly normal vision. Two views have been maintained as to their origin: (1) That they are hyaline excrescences of the lamina vitrea of the choroid which become embedded in the head of the optic nerve, and (2) that they have nothing in common with the choroidal excrescences, but are a special pathologic process confined to a small portion of the optic nerve. The microscopic studies of the author indicate that the latter view is the more nearly correct of the two. The exact nature of the material thus deposited has not been determined. One investigation by Hirschberg and Cirincione indicates that the bodies are amorphous and organic, and their composition appeared most to resemble that of elastin. They may undergo calcification, like the cheesy nodules in the lung. According to Parsons, "Drusenbildungen" upon the optic disk represent exudations which have been laid down in layers.

The affection is often termed *druses* of the optic nerve, but inaccurately, because they are outgrowths of the lamina vitrea; they may be massed together (see Fig. 175) in the macular region, or be scattered like small grains over the fundus, as in guttate choroiditis (see page 395).

CHAPTER XVI

AMBLYOPIA, AMAUROSIS, AND DISTURBANCES OF VISION WITHOUT OPHTHALMOSCOPIC CHANGES

"Amblyopia" and "amaurosis" are terms which signify *dimness of vision*, the former being used to describe *obscurity of sight*, and the latter the more advanced condition of *loss of vision*. Although these terms are often employed to indicate defective vision unexplained by lesions in the eye or refractive error, this limitation is not strictly followed, and eyes blinded by inflammatory disease are sometimes denominated *amaurotic*.[1]

Modern methods of examination have greatly lessened the number of conditions to which the older writers attached the words "amblyopia" and "amaurosis." Amblyopia is a symptom and describes the defective vision which the patient suffers. This may be due to functional disturbances or to disease of the visual apparatus (retina, optic nerve, or visual centers), and may be unassociated with changes in the eye-ground; or there may be atrophy of the optic nerve.

Amblyopia may be congenital or acquired; temporary or permanent; symmetric or non-symmetric.

Congenital Amblyopia.—This term is used to describe that variety of defective vision which for the most part is unassociated with fundus lesions, although sometimes the papilla is discolored and the macula deeply pigmented, and there is a scotoma, either small and absolute, or larger and for colors alone. According to Heine, a central scotoma can be demonstrated in 90 per cent. of the cases. The faulty vision has existed from birth, and often high grades of refractive error, especially hyperopia and astigmatism, are present, and clear images have never been focused upon the retina. Correction of the optical error usually fails to improve, materially, the vision; the retinal images continue to be defective. In very young patients an eye of this character may occasionally be trained to more nearly perfect vision after a proper correction of the refractive error, and this attempt should always be made.[2] Naturally, before the diagnosis of congenital amblyopia is established, the possible influence of orbital, nasal sinus, and central nervous disease must be eliminated. The origin of congenital

[1] The term "amaurosis" is also applied to certain cases of blindness in young children dependent upon hereditary influence, syphilis, tuberculous disease, and meningitis. The eye-grounds may or may not be diseased.

[2] A form of amblyopia has been described by Martin and called *astigmatic amblyopia*, dependent upon an imperfect development of the functions of the finer anatomic elements of the retina. It has been attributed to the fact that at the time of the education of the sense of sight, owing to astigmatism the retina has been asymmetrically stimulated, and consequently there has been asymmetry of visual acuteness.

amblyopia is obscure; an anatomic basis for the condition has not been discovered (Heine, Lohmann). Leber's tapetoretinal degeneration (page 538) is one cause of congenital blindness.

Defective vision, attributed to lack of use (*amblyopia ex anopsia, argamblyopia*, according to Gould), may occur on account of obstruction to the rays of light falling upon the retina—*e. g.*, congenital corneal opacities, congenital cataract, and impervious persisting pupillary membrane; or in an eye which from early infancy has squinted, and has, therefore, not been concerned in the visual act (compare with page 627). The amblyopia of a squinting eye may disappear if the seeing eye becomes blind or is removed; a number of such cases are on record.

Gould maintained that certain cases of amblyopia which have been attributed to disuse are really due to a low grade of choroidoretinitis affecting the macular region, brought into existence by an irritating stimulus with which a long-continued ametropia has supplied this area. It is probable that the explanation of many of such cases depends upon the presence of types of macular pigmentary degeneration (pages 508 and 538).

In this category of amblyopias are also placed certain congenital defects of structure—*e. g.*, coloboma of the iris and choroid and deficient development of the entire eye (microphthalmos). Retinal hemorrhages in the newborn explain some cases. Usually one eye is affected; if both are amblyopic, nystagmus commonly is present.[1] Occasionally children, usually under two years of age, are brought for examination with the history that following an illness characterized by convulsions, vomiting, and retraction of the head, sight had become exceedingly defective. The condition is a temporary one and usually recovery of vision is complete, being restored quickly or slowly, according to the severity of the original attack. Such *temporary amaurosis of infants* was described by Nettleship, Gay, and Stephenson. Among 60 cases of amaurosis in infancy collected by Foster Moore, B. T. Long, H. Neame, and P. G. Doyne, 5 of them were of this character. The etiology of this condition has been ascribed to distention of the third ventricle and pressure on the chiasm.

Congenital Amblyopia for Colors (*Color-blindness*).—Congenital disturbance of the color-sense has been found in about 3 per cent. of the examinations made for this purpose, but it is extremely rare in women (0.2 per cent.).[2] Both eyes, except in rare instances, are affected, and a distinct hereditary tendency has been noted in many instances. In other respects the functions of eyes which are "color-blind" are normal, and the cause of the condition has not been determined.

The methods of detecting color-blindness have been described on

[1] A persistent cramp of the lid, such as occurs in children, unrelieved for weeks at a time, may produce blindness, noticed when the eyes are finally opened, temporary in its character, with normal ophthalmoscopic appearances. In other cases the loss of vision, however, is permanent, with gross changes in the eye-ground. This condition has been referred to under Blepharospasm (page 194).

[2] For a study of "Color-blind Females and the Inheritance of Color-blindness in Man," by Ingolf Schiotz, see British Journal of Ophthalmology, Aug., 1920.

page 72. Congenital color-blindness must not be confounded with the various disturbances of the color-sense in diseases of the optic nerve and retina or in hysteria.

Derangements of the perception of colors have been divided into two varieties: the one characterized by an absence of the power to perceive colors, or *achromatopsia;* and the other characterized by difficulty in distinguishing colors, or *dyschromatopsia.* The former condition, or color-blindness, is rarely *total* as a congenital defect (a condition which is not uncommon as the result of pathologic changes in the optic nerve, etc.); generally it is *partial—i. e.*, one or more of the fundamental colors are not recognized.

According to Helmholtz's theory, three classes of *partial color-blindness* exist—blue-blindness (also called *violet-blindness*), green-blindness, and red-blindness—or, according to the new nomenclature, *tritanopia, deuteranopia,* and *protanopia.*

A person afflicted with *blue-blindness* (*yellow-blue blindness,* according to Hering) sees only red and green. He usually confounds blue with green, purple with red, orange with yellow, and violet with yellow-green or gray.

A person afflicted with *green-blindness* (*red-green blindness,* according to Hering), to quote from Thomson, confounds light green with dark red, does not recognize a dark green on black, but recognizes well a red one on the same background. Preyer states that the most frequent confusions are: brown with dark green, red with green, red with orange, red with yellow, red-yellow with green-yellow, bluish-green with purple.

A person afflicted with *red-blindness* (*red-green blindness,* according to Hering), again to quote from Thomson, confounds light red colors with dark green, and cannot see a dark red square on a black ground. According to Preyer, the most frequent confusions are: red with dark green, yellow with green, green with bright red, bluish green with gray, orange with greenish yellow or with red, orange with golden yellow, with grass green, or with red, purple with blue.

Red- and green-blindness are the most usual manifestations of color-blindness; it is often hereditary; the other type—blue-blindness —is not common. According to Lohmann, records concerning the hereditary factors in blue-yellow blindness are wanting. Knies has described congenital violet-blindness; red and purple are not distinguished from each other, both being called red.

In the second variety, or imperfection in the color-sense (reduced color-sense), the individual may correctly recognize brightly marked colors, but confuses colors which are closely allied and the various shades. To him violet and blue and orange and red are difficult distinctions. Dyschromatopsia should be distinguished from partial color-blindness (Landolt).

The *theory of color vision* has been the subject of much speculation, and many theories have been advanced, but none of them is entirely satisfactory. Two will be mentioned:

The *Young-Helmholtz theory* assumes the existence in the retina of three kinds of end-organs, each with its own photochemical substance, which can be decomposed by a certain color; that is, there is a red-sensitive substance, a green-sensitive substance, and a blue-sensitive substance. If a light mainly stimulates the red-, green-, or blue-sensitive substance it gives rise to the sensation, respectively, of red, green, and blue, while simultaneous stimulation of two or more of these substances gives rise to other color sensations, including white light. A color-blind person, according to this theory, is one in whom two of these substances have a like composition.

The *Hering theory* assumes the existence in the retina of a white-black, red-green, and yellow-blue visual substance, which may be either decomposed (disassimilated) or restored (assimilated) by the light. A destructive process, or one of *disassimilation*, in the white-black substance by white light or any other simple or mixed color, produces a sensation of white; a process of restitution, or *assimilation*, in this substance produces the sensation of black Red light produces disassimilation in the red-green substance, and thus the sensation of red; green light causes a process of restitution, or assimilation, in the red-green substance, and thus the sensation of green. From decomposition of the yellow-blue substance by yellow light arises the sensation of yellow, while the sensation of blue is produced by a process of assimilation in the same substance. A color-blind person, according to this theory, is one in whose retina the red-green or blue-yellow substance is absent.

Many objections to the Young-Helmholtz and Hering theories have been recorded and by some writers they are entirely rejected. Other theories have been propounded, notably one by Dr. Edridge-Green. It is not possible in a book of this scope to include them or to attempt an analysis of the subject. Those interested should consult "Introduction to the Study of Color-vision," by J. Herbert Parsons; "Color-blindness and Color-sense" and "Theories of Color-vision" in the American Encyclopedia of Ophthalmology, Vol. IV, 1914, and "Color-blindness," 2d Edition, by Edridge-Green, also "The Physiology of Vision" by the same author.

Treatment is ordinarily unavailing, but recent investigations indicate that if the defect is ascertained early enough systematic training may succeed in developing the deficient color-sense; hence the importance of the examination of the color-sense in young children.

Congenital Total Color-blindness.—This rare affection has been particularly well studied by Grunert and Uhthoff. To those affected (twice as many males as females) colors appear only as impressions of light and dark. According to Grunert, the colors at the red end of the spectrum seem lighter than to the normal eye, while those at the violet end seem darker. Total color-blindness is nearly always associated with defective central vision, nystagmus, and photophobia. The eye-ground may be normal; or there may be pallor of the disk and macular changes. A central scotoma is common. The eyes are more frequently myopic than hyperopic. Several members of the same family may be affected, and in some instances consanguinity of the parents has been determined.

Congenital Word-blindness.—In this condition the memory for the optic impression of words and letters is greatly deficient or wanting. The affection is more frequent in boys than in girls, although girls are by no means exempt, as has sometimes been stated. As C. J. Thomas points out, it may assume a family type, and in a number of instances more than one member of a family has suffered. Examination reveals normal eyes, good vision after any refractive errors have been corrected, and either inability to learn to spell and to read, or else great

difficulty in these respects. Sometimes figures are more readily recognized than letters, but *number-blindness* also occurs; sometimes the blindness is related only to letters (*letter-blindness*). In other respects the subjects of this affection are normal and other forms of memory are good; indeed, not infrequently the auditory memory is more developed than in a normal child. The condition is probably due to a congenital defect in the visual memory center for words and letters. As Hinshelwood (to whom we are particularly indebted for early studies of this affection) and all those who have written on the subject insist, great care should be exercised to detect this affection early in life, because it is much more common than is generally supposed. Its frequency is greater, in all probability, among the lower classes. Much can be done by systematic training—for example, with block letters—as Hinshelwood suggests, so that the child may assist the visual memory by the sense of touch. C. J. Thomas, discussing the treatment of this affection, suggests the phonic method as a suitable one to employ, because in it, at first at least, the visual word-images are ignored.

Reflex Amblyopia.—Certain cases of partial or complete loss of vision have been vaguely attributed to irritations in distant portions of the body—for instance, the presence of parasites in the intestinal canal. In many of these instances, however, a proper investigation has shown that other causes have been active in producing the defective sight.

A number of cases are on record in which an irritation transmitted through the branches of the fifth nerve has been supposed to produce an amblyopia, chiefly in association with disease of the teeth. At all events, in any case of amblyopia without ophthalmoscopic changes, and not readily classified in any of the well-recognized groups, a thorough examination of the teeth is advisable.

Traumatic Amblyopia.—This may occur after severe injuries of the head, especially in the occipital region and the region of the external angular process of the frontal bone; bruises along the course of the spinal cord after a railroad injury; and blows upon the brow in the region of the supra-orbital nerve.

In some of the cases there is a fracture across the optic canal, a hemorrhage into the intracranial cavity, or definite disorganization of the brain contents, followed by secondary changes in the optic nerve. (See also page 567.) In other instances no ophthalmoscopic changes are discovered, and the defective vision may be temporary in character, or there may be effusion or hemorrhage into the intersheath of the optic nerve, edema of the retina, and neuritis. *Hemorrhage into the sheath of the optic nerve* after fracture or traumatism of the skull has been observed a number of times and has been especially well studied by Uhthoff. In a specimen examined by the author and T. B. Holloway the sheaths were fully distended with blood and there were also many retinal hemorrhages. An extensive fracture of the base of the skull had occurred and had caused this *hematoma of the optic nerve-sheath.* Hemorrhage into the sheath of the optic nerve may have its source in an intracranial hemorrhage, for instance, rup-

ture of an aneurysm (F. H. Doubler and S. B. Marlow). Hematoma of the optic nerve-sheath was frequently observed during the past war following cranial and orbital injuries. In these cases peripapillary hemorrhage was comparatively rare, but a peripapillary brown ring due to hemic pigment was often observed. Atrophy of the optic nerve was the usual result.

Amblyopia after railroad injuries is often exaggerated by patients in the hope of securing damages.

During the recent war many soldiers developed in unusual degree a variety of nervous phenomena attributed to the concussions resulting from exploding shells, and the term *shell-shock* came into existence. Many of these cases were the result of a true neurosis



Fig. 256.—Hematoma of optic nerve-sheath (patient in the University Hospital).

which was neither conscious nor voluntary. Amblyopia was a common manifestation and varied from a reduction of vision which was described as "foggy" to complete blindness which was sometimes transitory, or short-enduring (several weeks), or long-enduring (months or even a year or more).

In general terms, the evolution of psychic blindness after a soldier was "shell-shocked" was as follows: During the period he was semi-conscious, or dazed, he was partly or entirely blind; vision often returned with the restoration of consciousness, or amblyopia persisted for varying periods of time, if the soldier's attention was fixed upon, or directed to, his eyes. By a process of autosuggestion the loss of vision was perpetuated. Associated with this psychic or hysteric cecity, there often was tonic blepharospasm, that is, a convulsive closure of the

lids, or clonic blepharospasm, that is, continued blinking of the lids, sometimes designated "fluttering" or "twinkling" of the lids. The pupils reacted normally; the eye-grounds were normal. The fields of vision, if it was possible to chart them, were variously contracted (see page 582). The restoration of vision was sometimes prompt; sometimes very deliberate. The "shooting eye" was often the last to recover.

The usual measures suited to the treatment of neurotics, suggestion, etc., were effective.

In a certain number of these cases of amblyopia following shell concussions the blindness depended upon an organic basis, that is, commotio retinæ, rupture of the choroid, intracranial lesions—a class very different from the one just described.

Amblyopia and amaurosis occur under the influence of disease and the toxic action of certain drugs, due either to a direct effect upon the retinæ and optic nerve, to an influence upon the visual centers, or to some change, perhaps of vasomotor origin, affecting the blood-supply of these structures.

In this category may be noticed:

1. **Uremic Amblyopia, or Amaurosis.**—This may develop in any form of renal disease, but is more common in the acute nephritis of the eruptive fevers, especially scarlet fever, and of pregnancy than in other varieties of kidney affections. In scarlet fever it appears with albuminuria in the stage of desquamation, and is bilateral, the blindness in many cases being absolute and often associated with brain symptoms: convulsions, vomiting, stupor, coma, and hemiplegia. In spite of the blindness, the preservation of the pupillary reactions is the rule; sometimes the pupils are dilated and motionless.

The ophthalmoscopic picture may be negative, or there is a slight neuritis, a little wooliness of the surface of the optic disk, or delicate edema of the retina. A *functional amblyopia* during *pregnancy* has neen noted, that is, one without the presence of albuminuria, perhaps due to toxemia or circulatory disturbances. The *prognosis*, as far as vision is concerned, is good.

The **treatment** does not differ from that which is applicable to the disease or condition which produced it.

2. **Glycosuric Amblyopia.**—In addition to the affections of vision already described in connection with diabetes (paresis of accommodation, premature presbyopia, alterations in refraction, cataract, retinitis, and retinal hemorrhages), there occurs an amblyopia in this disease in which the visual field is sometimes peripherally intact, sometimes peripherally restricted, and occasionally hemianopic, but in which there is a central color scotoma. Rönne ascribes diabetic amblyopia not to an interstitial neuritis, but to a process of degeneration. This amblyopia may be the only ocular symptom of diabetes, and in every case of amblyopia the urine and blood should be examined for sugar. It has seemed to the author that so-called glycosuric amblyopia is more apt to be found in diabetics who use tobacco freely than

in those who are abstainers in this respect. Should the scotoma develop the characteristics which have been described the amblyopia is probably due to tobacco (see also page 564).

The prognosis is unfavorable, and often the treatment, which should include the usual measures suited to diabetics, is not very efficacious.

3. **Malarial Amblyopia.**—In addition to amblyopias in malarial cachexia with lesions apparent in the fundus, are those cases, without such lesions, due to a special action of the malarial poison upon the optic nerve and the retina. There is transient loss of vision, or complete blindness, lasting from several hours to some days or even months. In most of the instances ophthalmoscopic findings are negative, or the descriptions are couched in vague terms applied to the retina and optic nerve—"congestion," "hyperemia," and "redder than normal." The affection may be unilateral or bilateral. It is quite possible that large doses of quinin may have been responsible for some of the types of so-called malarial amblyopia.

4. **Amblyopia from Loss of Blood.**—Loss of sight often follows hemorrhage, more frequently if this is spontaneous than if it is traumatic, and is said to be most complete after hemorrhage from the stomach. It also may follow epistaxis, hemoptysis, urethral, uterine, and intestinal hemorrhage.

Two very different results may ensue: Either a temporary blindness, owing to the impoverished blood-supply of the visual centers or retina, or a permanent loss of sight and atrophy of the optic nerve. Ward Holden has shown that the amblyopia following hemorrhage is due to degeneration of the retinal ganglion-cells, together with their long processes, which make up the centripetal fibers of the optic nerve.

The ophthalmoscopic appearances vary from a slight pallor to complete atrophic whiteness of the papilla, with contraction of the arteries. The lesions in the unfavorable cases usually do not appear until a week or more after the hemorrhage has taken place. Optic neuritis and hemorrhages into the retina may also arise. Occasionally, the papilla is highly edematous, suggesting in appearance a small white mound, the so-called "pallid edema of the disk." In a patient recently under the author's care this phenomenon was most marked, the swelling attaining a height of about 5 D. Hemorrhage following criminal abortion was the cause; blindness was temporarily practically complete; partial restoration of vision occurred. The prognosis is most favorable in uterine cases.

The **treatment** consists in the use of iron, arsenic, and strychnin, complete rest, and an easily assimilated diet. Intravenous saline injections are also recommended as remedial agents (Elschnig).[1]

Amblyopia from the Abuse of Drugs.—A certain number of toxic agents (lead, tobacco, alcohol, etc.) produce an axial neuritis

[1] Sudden blindness with preserved pupillary reaction and without ophthalmoscopic changes has been noted in whooping-cough, and is probably due to edema between the corpora quadrigemina and occipital lobes. Sudden blindness in old persons with arteriosclerosis has been observed (see also page 567).

or a degeneration and destruction of the retinal ganglion-cells (see page 566).

Amblyopia, more or less complete, may also arise under the toxic influence of nitrate of silver, chlorate of potassium, mercury, arsenic, atoxyl, bisulphid of carbon, nitrobenzol, salicylic acid, oil of wintergreen, cannabis indica, coffee, tea, stramonium, male fern, iodoform, osmic acid, chloral, antipyrin, and lead (see also page 565). The last agent may produce a neuritis, but also an amblyopia without ophthalmoscopic changes. It is usually transient, occurs in acute cases, and was compared by Gowers to the temporary amaurosis of uremia.

Some of these toxic agents are responsible for a central scotoma—for example, bisulphid of carbon, stramonium, and iodoform; complete blindness and atrophy of the optic nerve may be the result of the action of others—for instance, male fern and iodoform. Dinitrotoluene-amblyopia may occur among munition workers and amblyopia is also due to nitrophenol. Associated conditions are peripheral neuritis. The amblyopia, if caused by explosive substances, for example, dynamite, has been attributed to the inhalation of the vapor (Brose). In some of these cases the influence of tobacco cannot be excluded (*T. N. T.*, for instance [T. M. Legge]). Ocular disturbances caused by carbon monoxid are noteworthy (combustion of carbon-containing material in the production of water gas, in the exhaust of an automobile, burning coal, etc.). Headache, blurred vision, and even optic-nerve atrophy have been described by Wilmer.

The loss of vision which occurs under the influence of four substances—quinin, ethylhydrocuprein, methyl-alcohol, and atoxyl—deserves special mention.

Quinin Amaurosis.—Although in most instances *quinin amblyopia*, or *amaurosis*, follows the ingestion of a large quantity of the drug, occasionally the symptoms are caused by moderate doses. The author has seen 12 grains (0.78 gm.) produce decided temporary amblyopia in a susceptible and neurotic woman.

The characteristic clinical features of quinin amaurosis are total blindness subsequent to taking large doses of the drug, extreme pallor of the optic disks, marked diminution of the retinal blood-vessels in number and caliber, and contraction of the field of vision. Other symptoms which have been noted are: diminution of the color- and light-sense, dilated pupils, and immobile iris during the blind stage, and occasionally anesthesia of the cornea. Usually the effect of quinin upon the ear is manifested by deafness and tinnitus.

The restoration of central vision may be perfect or incomplete. The contracted field of vision gradually widens out, but does not regain its normal limits. The disk may remain pallid and atrophic in appearance years after the poisoning; in other instances it resumes its normal tint, but usually the contracted vessels do not regain their proper caliber. Reduction of light-sense is a permanent feature. In one case (Gruening) a cherry-colored spot was noted in the macula,

in another a scotoma in the visual field. Occasionally the blindness is permanent.

The blindness following the administration of toxic doses of ethylhydrocuprein (optochin), especially in the treatment of pneumonia, resembles in all respects that of quinin amblyopia.

The first effect of the toxic influence of quinin is to lessen the blood-supply of the retina and optic nerve, and later, as the author has experimentally shown in dogs, permanent optic-nerve atrophy ensues. Ward Holden has demonstrated, and his results have been fully confirmed by Drualt, Birch-Hirschfeld, and a number of other observers, that the blindness is due to a degeneration of the ganglion-cells and nerve-fibers of the retina, followed by an ascending degeneration of the optic nerve. Anatomic examination of eyes blinded by optochin reveals degeneration of the ganglion-cells, vascular change, and partial atrophy of the disk (G. Abelsdorff).[1]

The **treatment** consists in the administration of nitrate of amyl, which temporarily improves vision, and of the exhibition of strychnin and digitalis.

Methyl-alcohol Blindness, or Amaurosis.—The amount of wood alcohol which may cause blindness represents a varying quantity. Thus, blindness and atrophy of the disk have followed the ingestion of 2 to 5 drams (7.8–19.4 gm.), while recovery after drinking ½ pint (236 c.c.) of this liquor has been observed (Moulton). In short, methyl-alcohol intoxication is an example of idiosyncrasy (F. Buller, C. A. Wood). The number of immune persons, however, cannot be great. Methyl-alcohol itself, Columbian spirits, other varieties of purified wood alcohol, and the drug as a substitute for ethyl-alcohol in cheap whiskies and other alcoholic beverages, as well as in Jamaica ginger, certain essences, bay rum, cologne water, etc., are capable of producing most violent general toxemia and visual disturbance. Igersheimer believes the influence of methyl-alcohol depends upon the admixture of fusel oil. Briefly, the symptoms are these: Intense gastro-intestinal disturbance if the dose is not too large, followed, if it is greater, by severe headache, giddiness, and coma; rapid failure of sight, which may improve, but soon relapses; contracted visual fields and usually absolute central scotomas; and, finally, total or nearly total blindness. Ophthalmoscopically, blurring of the edges of the disk, positive neuritis (rare), and complete atrophy without signs of preceding inflammation have been observed. In many instances there is diminution in the size of the retinal vessels. Occasionally there is decided pain on movement of the eyes or on pressing them backward into the orbit. The *prognosis* of methyl-alcohol poisoning is most unfavorable. A number of fatal cases have been reported. Not only may the poison enter in the usual manner through the stomach, but blindness has resulted from inhalation, aided by absorption, as the author has shown, through the cutaneous surface.

[1] The most recent and complete review of all of the manifestations of quinin blindness is by R. H. Elliot. See American Journal of Ophthalmology, September, 1918.

A few examples of restoration to nearly normal vision have been reported. The blindness is attributed by Ward Holden, Birch-Hirschfeld, and Schwarzkopf to nutritive changes in the ganglion-cells of the retina.[1] It is possible that there may be a simultaneous action on the ganglion-cells and the tissues of the optic nerve (Gifford). The *treatment* should include diaphoresis, emetics, the administration of iodid of potassium, and later strychnin. S. Lewis Ziegler highly recommends the exhibition of alkalies to neutralize the acidosis which is present in early stages of this toxemia; also negative galvanism applied directly to the eye. Lumbar puncture has been recommended and favorable results have been reported.[2]

Arsenic Amblyopia.—Atoxyl has been much employed in the treatment of various conditions, notably certain skin diseases, chlorosis, syphilis, and trypanosomiasis. Serious visual disturbances have followed its use in a number of instances. The following have been reported: Reduction of visual acuteness from one-half to complete blindness; contraction of the visual field, especially on the nasal side; pallor and atrophy of the optic disk, with narrowing of the retinal vessels; usually no central scotoma, but at times a central scotoma for colors and sometimes visual hallucinations and colored vision (cyanopsia). In one patient retinal hemorrhages were found. The amount of the drug which has produced these visual disturbances has varied considerably: 1.2 grams, given subcutaneously for anemia, within twenty-six days (Steinebach); 5.1 grams within twenty-six days (Lesser and Greeff); 50 grams within seven months (von Krüdener); 4.5 grams within one month (Kopke). Koch observed a number of cases of blindness without ophthalmoscopic change after injection of 1 gram of atoxyl for the cure of sleeping-sickness. The blindness has been ascribed to optic-nerve atrophy with primary involvement of the retina, and to retrobulbar neuritis and consecutive atrophy; it has also been attributed to central lesions.

Blindness following the administration of other *arylarsonates*, for example, soamin, arsacetin, hectin, orsudan, has been reported. The harmful action is due to the anilin in these compounds and not to the arsenic. Amblyopia due to inorganic arsenic compounds presents features entirely different from those which the organic preparations produce, and the prognosis is good (Schirmer). The arylarsonates

[1] In Ziegler's excellent study of the ocular menace of wood alcohol poisoning (British Journal of Ophthalmology, 1921, v, p. 365) the suggestion appears that a disorder of the pituitary body should be regarded as the primary and fundamental lesion of this toxemia. This was not confirmed by a research (dogs were used) of the author, Alexander Fewell, and P. De Long.

[2] The retinal ganglion-cells of dogs acutely and chronically poisoned by the administration of commercial methyl-alcohol (researches of the author, Fewell and De Long, and of J. Friedenwald and Felty) failed to show even the slightest departure from normal. Also there was no evidence of blindness or of ophthalmoscopic change. In Schwarzkopf's experiments the animals employed exhibited no ophthalmoscopic lesions in the retina or optic nerve, but microscopically he found degenerated retinal ganglion-cells, thus confirming Birch-Hirschfeld's results.

cause progressive optic-nerve atrophy. It has been suggested that the primary action of the drug may be on the blood-vessels (E. T. Collins). The harmful dose has varied; much depends on idiosyncrasy. Salvarsan does not cause blindness, *i. e.*, it has no poisonous influence in proper dosage on the healthy optic nerve.

Hysteric Visual Disturbances (*Amblyopia, Amaurosis, Asthenopia*).—Hysteric amaurosis is characterized by complete abeyance of the visual sensation. It occurs both as a unilateral and a bilateral affection, the former being far more frequent than the latter. The subjects of this condition are more commonly females than males. Occasionally, the blindness lasts but a very brief period of time, and occurs during a crisis; at other times it lasts for weeks, months, and, it is said, for years. The eye-grounds are normal. Usually the pupils react to the influence of light. Sometimes only a feeble contraction follows the light stimulus; occasionally the pupils are dilated and insensitive to light (Kerneis). Generally it is possible to prove by ordinary prismatic, stereoscopic, and other tests that the supposed blind eye really sees. The exact similarity of these phenomena and those associated with war neuroses (so-called "shell-shock blindness") is evident (see page 576).

In place of amaurosis, incomplete anesthesia of the visual sense, or hysteric amblyopia, may occur. This includes reduction of visual acuteness, disturbances of the visual field for white and for colors, dischromatopsia, and achromatopsia. The visual field in hysteria is characterized by concentric contraction, which is evident at the beginning of the examination, and is not produced by repeated measurement (retinal tire field), and the amount of reduction varies from a slight contraction to such extreme restriction that the most peripheral circle is just beyond the fixing-point. Sometimes the field has a *tubular* character—that is, the contracted visual field maintains the same size, no matter at what distance from the examined eye the point of fixation is placed. Similar reductions take place in the field for colors. A somewhat characteristic variation is that the red field is the last to be affected, with the result that its extent may exceed that of blue, and become the most peripheral of the color circles. Occasionally it is the most peripheral circle for the entire field. This is the so-called *inversion of the color-field*. Sometimes there is an excessive extent of the color circles. A rare hysteric phenomenon is central scotoma; zonular scotoma and the so-called oscillating field have been observed.

The visual field phenomena in hysteria are, according to Babinski, due to suggestion. If suggestion can be eliminated they do not develop. Morax, at first impressed as most observers were, with the significance of the retracted field of vision in hysteria, has adopted Babinski's views. Believing that it is impossible to avoid suggestion during ordinary perimetric examinations, he relies upon simpler methods, taking the field with the aid of outstretched fingers or other familiar objects. Hurst and Symns as the result of their studies of war neuroses reach the same conclusion.

In the light of our present-day knowledge there is no question that too great stress has been laid on concentric contraction of the visual field and inversion of the color lines as stigmata of hysteria. None the less these examinations are interesting and valuable in the study of such cases from the very fact that they demonstrate, or help to demonstrate, the presence of the abnormal nervous mechanism of highly suggestible patients.

It should be remembered that inversion of the color-fields is not peculiar to hysteria; it has been observed in brain tumor, ataxia, hemorrhage in the brain, and in certain toxemias, notably those produced by lead and nitrobenzol. *Crossed amblyopia*—that is, complete or partial blindness on the same side as the hemianesthesia, and associated with some deficiency of acuteness of vision upon the opposite side—is sometimes an hysteric manifestation. Hemianopsia in an enduring form is never due to hysteria. As a temporary visual-field phenomenon it has been observed.

Hysteria produces many other functional disturbances of the eye—monocular diplopia, ptosis, blepharospasm, conjugate deviation of the eyes, and the great symptom-group gathered under the term "retinal asthenopia" (see page 482).

The **prognosis** of these cases in the main is good, although the apparent blindness may last for long periods of time.

The **treatment** consists of measures calculated to improve the condition of the patient—massage, rest, electricity, and tonics. Usually a cure can be secured by suggestion.

Pretended Amblyopia (*Malingering*).—For the purpose of escaping irksome duties—for example, in the army—or to excite sympathy patients occasionally pretend to be blind in one eye. In order to detect the deception many plans have been originated. Three methods will be described:

1. **The Diplopia Test.**—This is performed in the same manner as the ordinary examinations of the exterior ocular muscles with prisms. The subject is seated before a lighted candle at 20 feet distance, and a 7° prism placed before the admittedly sound eye. If, now, superimposed double images are acknowledged, there is binocular vision, and the fraud is detected. The examiner may vary the test by placing the prism before the supposed blind eye, either base up or base down. A prism of 10° base outward may be placed before the eye for which blindness is claimed. If this eye sees, double vision will be produced and the eye will move inward to correct it and fuse the two images.

2. **Harlan's Test.**—This is an extremely useful and simple test, and is performed as follows: Place an ordinary trial-frame upon the subject's face and put before the admittedly sound eye a high convex glass (+16 D), and before the eye which is claimed to be blind a plane glass or a weak concave spheric (−25 D), which will not interfere with vision. If letters placed at a distance of 6 meters are read, the act of reading must have been done by the eye which was claimed to be

sightless, inasmuch as vision at that distance with the other eye is excluded by the presence of the high convex lens. The test may be further elaborated by covering the pretended blind eye and requesting the patient to read the letters; if he is unable to do so, the fraud is at once exposed.

3. **Tests with Colored Glasses and Letters.**—The method generally employed, or some modification of it, is known as *Snellen's method.* The patient is required to look at alternate red and green letters. The admittedly sound eye is covered with a red glass, and if the green letters are read, evidence of fraud is present. Instead of a red glass, a green glass may be used, through which the red letters will be invisible. Ingenious letters, based upon the fact that red upon a white background viewed through a red glass disappears, and viewed through a green glass appears black, have been designed. Tests with stereoscopes may also be employed to detect malingering.

If a malingerer claims to be blind in both eyes, these tests will not avail, and he can be detected by placing a careful watch over him. The fact that the pupil contracts on exposure to light does not prove that there is sight in the eye, because, as Swanzy pointed out, a lesion in the center of vision, or in the course of the fibers connecting this center with the corpora quadrigemina, producing absolute blindness, would still permit a perfect reaction of the pupil to light. Priestley Smith and E. Jackson suggest the following test for feigned *binocular blindness:* Place a lighted candle in front of the subject; now hold a 6° prism, base out, before one eye; if both eyes see, the one behind the prism will move inward, and on removing the prism will move outward, the other eye remaining fixed.

Night-blindness (*Functional Night-blindness; Often Incorrectly Termed Hemeralopia, but Properly Nyctalopia*).—It has already been pointed out that night-blindness is one of the early symptoms of pigmentary degeneration of the retina. In the present condition, however, there are no retinal lesions.

It is a functional defect of the retinal apparatus concerned with dark adaptation (Treitel, Birch-Hirschfeld) which may be and often is congenital. Determining causes are exposure of the eye to strong light and glare, together with a debilitated and often scorbutic state of the system, defective nutrition, autotoxemia, etc. Other causes are diet deficient in fat and albumin, disease of the liver, malaria, and alcoholism. It affects residents in tropical countries, often soldiers and sailors, and has been occasionally observed in large schools, usually in the early spring or summer (Nettleship, Snell). It has been reported as an endemic in certain countries, especially in Russia during the Lenten fasts.

During the past war numerous cases of night-blindness were discovered in which the determining causes already named were operative; also great fatigue, loss of blood, refractive error, especially myopia and astigmatism. Not infrequently a history of previous defective night vision was obtained; often the eye-grounds were normal. Sometimes

they showed abnormal pigmentation. The visual symptoms were those which have been recorded, especially reduction of vision in diminished light and constricted visual fields, particularly for blue. A noteworthy group was composed of those men who became conscious of their defective sight because for the first time in their lives they were forced to live a nocturnal life and could contrast their vision with that of other men who were normally sighted. Almost invariably they were ametropes. They have been aptly described by M. Landolt as *nocturnal amblyopes*. Naturally many soldiers were found who did not belong to the "functional" group, but were night-blind because they were the subjects of chorioretinitis and pigmentary degeneration of the retina which was not discovered when they entered the service. Augstein has described a fundus appearance, the so-called whitish-gray fundus, which he attributed to decoloration of the pigment epithelium.[1] Naturally, a large number of those who claimed to be "night-blind" were malingerers.

Krienes divides the affection into *acute essential nyctalopia* (hemeralopia) and *chronic nyctalopia*, and he gives the following syllabus of symptoms: Decided dread of light, abnormal width of the pupil in the dark, depreciation of the central quantitative color-sense, particularly the blue sense in daylight, narrowing of the color-fields in daylight, particularly the blue field, abnormal shrinking of the visual field for white and colors in increasing twilight. Other not absolutely constant symptoms are loss of visual acuteness by daylight, shrinking of visual field for white in daylight, retinal tire field, paresis of accommodation, epithelial xerosis, erythropsia, and xanthopsia (see also page 254).

Night-blindness is occasionally a *family disorder*. Bordley has described a negro family of night-blind persons extending over five generations. The subjects eventually became blind, and shortly after blindness death ensued. Nettleship has published a history of stationary night-blindness in nine consecutive generations.

Treatment.—This includes the administration of iron, quinin, strychnin, and cod-liver oil, according to the indications. Dark-colored glasses should be worn. If scurvy is present, the diet and remedies suited to this condition should be prescribed. Suitable diet is important in all cases. Refractive error should be corrected.

Day-blindness (*Often Incorrectly Termed Nyctalopia, but Properly Named Hemeralopia*).—This is an affection, or rather a symptom, as the name implies, characterized by the fact that its subjects see better on dull days and in the dark than in a bright light. The visual field is not concentrically contracted.

This symptom occurs with the condition described by Arlt as *retinitis nyctalopia*, and with orbital optic neuritis of the chronic type (tobacco amblyopia, see page 564). It also occurs in other affections

[1] Consult "Night-blindness in Warfare," by Birch-Hirschfeld, Arch. f. Ophthal., vol. xcii, pt. II, 1916, and "Nocturnal Visual Defects Among Soldiers," by Marc Landolt, Arch. d. Ophthal., July–Aug., 1917.

of the optic nerve and in some diseases of the retina. The same condition may be present in certain congenital anomalies—albinism, coloboma of the iris and choroid, and irideremia.

It may develop in those who have long been excluded from the light. It may be congenital, and may be associated with an amblyopia of like origin.

A tonic **treatment** should be tried and the retina gradually educated to endure bright light.

Snow-blindness.—As this ordinarily is seen in northern regions, it is an affection of the conjunctiva. There are burning pain, photophobia, blepharospasm, hyperemia of the conjunctiva, and chemosis. In severe cases there may be ulceration of the cornea. The pupils are small, and there is congestion of the retina. The visual acuteness may be unaffected, or it may be distinctly lessened, especially if corneal complications arise; under these conditions restricted visual fields, both day- and night-blindness, red-green blindness (Lohmann), and scotomas have been observed. The local irritation in snow-blindness is analogous to sunburn, and like it has been attributed to the action of ultraviolet rays (Widmark). The pain caused by *glare* is due to excessive contraction of the orbicularis and to pressure of the supra-orbital nerve against the frontal bone.

Symptoms analogous to so-called "snow-blindness" develop as the result of exposure of unprotected eyes to any type of light rich in ultraviolet rays—for example, a naked arc light (*electric ophthalmia*); molten metal in electric welding; the flash of a short-circuit high-tension current, and a mercury vapor lamp (see also page 454). To the *injurious effect of light* in these circumstances, interpreted by the irritative phenomena already described, Parsons has applied the term *photophthalmia*. Although the ultraviolet rays probably cause the greater damage, rays of greater wave-length—infra-red and luminous rays—are also capable of harmful action. For a long time after subsidence of photophthalmia there may be persistent *asthenopia*, ciliary pain, frontal and other placed headaches. Those who are much engaged in work with roentgen rays often acquire decided conjunctival hyperemia or positive conjunctivitis (*x-ray conjunctivitis*).

Experimentally, it has been demonstrated that ultraviolet rays may cause changes in the lens (Hess) and in the retina (Birch-Hirschfeld). These changes are not likely to occur under ordinary conditions in healthy persons. The lens protects completely the retina of the normal eye (Verhoeff and Bell). Special forms of glass supposed to have the power of absorbing ultraviolet rays have been devised; all of them have a greenish-yellow tint, chief among them are "Enixanthos," Fieuzal glass, and "Euphos" glass. Crookes' glass (light and dark shades) is very satisfactory, and is much employed. According to Parsons, almost colorless didymium glass is the best protection against ultraviolet rays. The chief usefulness of protective glasses, according to Verhoeff and Bell, is not so much in their absorption of

any specific radiations, as in their effect in reducing the total amount of light so that it ceases to be dazzling.[1]

Chromatopsia.—Color vision in glaucoma (iridescent vision), in the form of variously tinted halos about the lamp-lights, has been described, and patients with blind eyes occasionally complain of being conscious of colored lights, owing probably to irritation of the visual centers.

Erythropsia in most instances has been noted after cataract extraction. Visual acuteness is not affected, but everything appears of a red or violet color. According to Fuchs, erythropsia can be caused by the visible rays alone. Verhoeff and Bell deny that ultraviolet rays are concerned with the production of erythropsia, which they believe is merely "a special case of color fatigue." It also occurs during ordinary mydriasis and in eyes which attentively regard a brilliantly illuminated surface. A not very uncommon phenomenon is *blue vision* or *cyanopsia*. *Green vision*, or *chloropsia*, has been noted after cataract extraction and corneal wounds, and in connection with diseases of the optic nerve and retina—for example, tabetic optic-nerve atrophy. *Yellow vision*, or *xanthopsia*, occurs in jaundice; it also has been noted as a symptom of santonin- and digitalis-poisoning. Bromid of potassium is said to be of service in ameliorating the chromatopsia which follows cataract extraction.

Micropsia and **macropsia** have been described in connection with syphilitic retinitis. They may appear as functional disorders in hysteric cases.

[1] "Some Effects of Bright Light on the Eyes," by J. Herbert Parsons, Trans. of Section of Ophthalmology, Amer. Med. Assoc., 1910. For a full consideration of this subject consult "The Pathological Effects of Radiant Energy on the Eye," by F. H. Verhoeff and Louis Bell, Proceedings of the American Academy of Arts and Sciences, vol. 51, No. 13, July, 1916.

CHAPTER XVII

AMBLYOPIA OF THE VISUAL FIELD, SCOTOMAS, AND HEMIANOPSIA

The importance of perimetric measurements in the study of various forms of ocular disease, especially in glaucoma and in affections of the retina, choroid, and optic nerve, has been noted (for the methods of examination consult Chapter II). There remain to be considered certain conditions in which a defect in the field of vision constitutes one of the most prominent symptoms.

1. **Partial Fugacious Amaurosis** (*Flimmer-scotom; Scintillating Scotoma; Migraine Ophthalmique*).—The symptoms are: A sense of vertigo; a positive darkening of the field of vision of each eye, beginning at the center and widening out in a vibratory movement until it overspreads the field, with corresponding sinking of the central acuteness of sight; or a scotoma surrounded by flashing zigzag lines ("fortification spectra") and cessation of the amaurosis with the onset of headache and vomiting; *scintillating scotoma* and a *transitory hemianopsia* without light or flashing are also observed. These symptoms may be, and often are, a prodrome of hemicrania or of true *migraine*, but they also arise without it. Scintillating scotoma in its various manifestations is very common not only in true migraine, but in association with other conditions, for instance, the menstrual epoch with or without hemianopsia, which is homonymous. Migraine and its visual phenomena are attributed by J. Herbert Fisher to temporary disturbance of the pituitary body. Cases of migraine occur which are preceded by apparitions of unusual character in place of the ordinary scintillating scotomas—figures of men, women, animals, etc. The condition probably depends upon circulatory disturbances in the occipital lobes or upon vascular spasm. Occasionally the *hemianopsia of migraine* after long periods of time and repeated attacks becomes permanent.

The **treatment** is concerned with the headache, the partial amaurosis being exceedingly temporary in character, and includes measures calculated to relieve migraine. The following drugs are of service: ammonium benzoate, bromid of potassium and caffein, extract of cannabis indica, and aspirin.

2. **Amblyopia of the Visual Field** (*Anæsthesia Retinæ*).—This functional disturbance as part of a general neurosis has been described on page 482. Because of the peculiar changes in the visual field many authors prefer the name "amblyopia of the visual field" to that of "anesthesia of the retina."

Fatigue restrictions of the visual field in the form already described are seen after injuries (*traumatic neurosis*), and sometimes with *traumatic anesthesia of the retina*. The element of hysteria cannot always

be eliminated, and the phenomena described in connection with hysteric ocular manifestations may predominate.

3. **Scotomas.**—Any lesion which blots out the function of a portion of the retina produces a corresponding blind area in the field of vision, or a scotoma—for example, a hemorrhage, a patch of retinochoroiditis in the macular region, or spots of disseminated choroiditis in the periphery of the eye-ground—which may or may not be positive. In rare instances the scotoma seen by a patient with central retinochoroiditis is colored. Papillitis causes an enlargement of the natural blind-spot, and retrobulbar neuritis a central scotoma. The different forms which scotomas assume are described on page 91. The scotomas associated with chronic glaucoma are depicted on page 419. Unilateral scotomas may occur in hysteria, in neurasthenia (central exhaustion scotomas), with menstrual disorders, in obstruction of the central artery of the retina, and with disease of the macular cortical center. *Ring-shaped* or *annular* scotomas have attracted much attention. Ordinarily, they are to be explained by the presence of chorioretinitis, but the statement, often made, that they always are of retinal origin is not correct. They interpret certain lesions of the optic nerve, and in addition to those caused by chorioretinitis and pigmentary degeneration of the retina (page 506), are those which occur with optic neuritis, choked disk, sinusitis (ethmoid and sphenoid disease), and chronic glaucoma (see page 420). They have also been observed in hysteric subjects and have been reported as part of the symptomatology of migraine (Zentmayer).

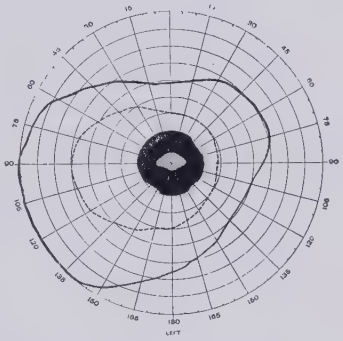


FIG. 257.—Ring-shaped scotoma in a case of optic neuritis.

In addition to these diseases certain affections of the optic nerve and of the chiasma are accompanied by a scotoma.

(*a*) **Central Amblyopia with Scotoma** (*Toxic Amblyopia*).—This affection has been described on page 564.

(*b*) **Chiasmal Central Amblyopia.**—Central scotomas, difficult to distinguish from those occurring in toxic amblyopia, are sometimes the initial signs of chiasm disease. According to Nettleship, the loss of the central field in the earlier stages is more abruptly defined than in tobacco amblyopia. With increase in the size of the growth they may expand into complete bitemporal hemianopsia (see page 594). Although the evidence is as yet insufficient, it is probable that disseminated sclerosis is a more common cause of chiasmal interference than is generally recognized.

As the visual phenomena in pituitary body disease have assumed such importance in ophthalmic and neurologic examinations, a brief résumé of them follows:

Visual Phenomena in Pituitary Body Disease.—These consist in impairment of vision, varying from blurred sight to complete blindness; intra-ocular optic-nerve alterations, varying from partial or general pallor of the nerve-head to partial or complete atrophy, less frequently postneuritic atrophy, choked disk, and non-prominent optic neuritis; alterations in the visual fields for white and colors, especially homonymous and bitemporal hemianopsia, and scotomas. Other ocular symptoms which have been described are visual hallucinations, chromatopsia, especially cyanopsia, persistent photophobia, palsy of various exterior ocular muscles, nystagmus, exophthalmos, and thickening and pigmentation of the eyelids with hypertrophy of the palpebral glands. A very early symptom, noticeable sometimes for weeks and months before fundus changes are detected, is a form of blurred vision, characterized by ill-defined indistinctness of sight. Visual disturbances are more frequent in primary hypopituitarism than in acromegaly. Generally the ophthalmoscopic appearances are those of so-called simple atrophy; often the disk has a characteristic waxy appearance; atrophic cupping is not evident; choked disk is rare. Even where the pallor of the disk gives every indication of simple atrophy, marked improvement in vision may follow successful operation or prolonged organotherapy or radiation (radium, *x*-rays). Evidently there is a physiologic block to light impulses, and not at this period a destruction of the nerve-fibers which is evident when shrinking of the nerve-head begins. While bitemporal hemianopsia is the most common visual field defect, homonymous lateral hemianopsia is not infrequent (about 6 per cent. in the author's experience), especially if with homonymous defects are included those alterations which Cushing calls the tendencies in this direction. Defect for colors precedes that for white, but if the visual fields are measured with small test-objects (page 87) the early changes can be detected without recourse to color tests, which often are unsatisfactory. There may be unilateral hemianopsia, or one eye blind and the temporal field of the other eye defective or lost—a frequent condition. In bitemporal hemianopsia in this disease it will be found that the temporal field tends to be lost from above downward; it represents an advancing process and may be divided into periods or "stages." The deformation proceeds on the temporal side of the field from above downward and from below upward in the nasal field or, to use Traquair's neat description, it "progresses round the fixation point clockwise in the right field and counterclockwise in the left." The final stage of this advancing process is most frequently attained in the upper nasal quadrant (about twice as often as in the lower [Traquair]). Naturally, there are many departures from this type, and a peculiarity of the visual fields in pituitary body disease is the variations which they undergo, hence the necessity of frequent perimetric observations.

Central and *paracentral scotomas* are common, especially the paracentral varieties; they may be situated to the outer side of each fixation point, that is, they are bitemporal hemianopic scotomas. These scotomas may antedate the more elaborate visual field defects and they

may expand into complete hemianopsia. In typical cases their behavior, that is, their progress is comparable to that of the general field. Early changes are found above the blind-spot; indeed, the upper outer part is the weakest part of the field. For a correct interpretation of pituitary body disease the visual fields must be frequently examined with test-objects of various sizes (page 87). As Traquair points out, relative bilateral scotomas may occur in disseminated sclerosis, and the centrocecal scotoma of pituitary body tumor has been mistaken for toxic amblyopia. Proper analysis of the scotoma, as stated, should prevent a mistake in diagnosis. The search for scotomas is important, as is their interesting relation to later visual field defects, and their character is revealed by careful analysis with the aid of small test-objects. They were first properly described by Foerster and have been studied by many observers (Bartels, Uhthoff, Rönne, Traquair, Jofenson, Walker, Holloway, and the author). They have demonstrated the necessity of frequent examination of the visual fields with test-objects of various sizes.

(*c*) **Stationary Optic Atrophy with Scotoma.**—This condition Jensen has described as one characterized by a scotoma similar to the one which occurs in toxic amblyopia, but much more decided. There are, according to him, marked diminution of central vision, a depreciation of the color-sense, and ophthalmoscopically the appearances of optic-nerve atrophy. The process is stationary, and vision does not improve under treatment. Jensen finds this affection exclusively in men before their forty-fourth year. It has a hereditary tendency, and is said to be caused by exhaustion and lack of sleep. Sometimes no cause can be demonstrated. Preceding the atrophy there may be slight neuritis. Hereditary optic neuritis with central scotoma (*Leber's disease*) has been described on page 561

(*d*) **Progressive Optic-nerve Atrophy with Scotoma.**—This includes the cases in which the optic-nerve atrophy of spinal disease (tabes dorsalis) is associated with a scotoma. The scotoma may be central or may be like the one in tobacco amblyopia (centrocecal), but as the disease progresses the peripheral field contracts, and finally it becomes difficult to detect the central defect. A central scotoma in tabetic atrophy of the optic nerve, according to Fuchs, is of more frequent occurrence than the ordinary records would seem to indicate. The scotoma is nearly always bilateral, and he regards it not as an accidental complication, but as an integral part of the tabetic process. Some authors maintain that the influence of tobacco and alcohol in the formation of these scotomas cannot be eliminated.

Central scotoma is common in insular sclerosis. Traquair describes the following features and behavior of the scotoma as suggestive: Rapid onset, changeable and fluctuating course, terminating usually in complete or nearly complete recovery. The focus may be situated in the chiasmal termination of the nerve (Velter), in the chiasma (page 589), and in the optic tracts. He considers central unilateral hemianopic or quadrant defect (junction-scotoma) or bitemporal or homonymous

hemianopic scotomas without peripheral field defect and of a fleeting or wandering character as suggestive. When the lesion is below the termination of the nerve the central scotomas vary in type and have no distinctive features.

(*e*) **Optic Neuritis with Scotoma.**—An unusual symptom of intraocular neuritis caused by meningitis is a central scotoma, either relative or absolute. The student should not confuse this with an enlargement of the natural blind-spot due to papillitis or papilledema. Ring scotoma may be associated with choked disk caused by brain tumor, and in optic neuritis due to sinusitis a central scotoma may develop (see also page 678).

Obscuration of One-half of the Visual Field, or Hemianopsia.[1]—In diseases of the eye—*e. g.*, glaucoma—one-half of the visual field may be wanting, and also in optic-nerve atrophy and neuritis, even if they are unconnected with disease of the visual pathway. These cases, however, are not included in the present account.

Hemianopsia is that defect of vision characterized by an obscuration, or loss of one-half of the field, which occurs under the influence of a lesion situated at the optic chiasm, in the optic tract, in the visual radiations, or at their ultimate destination in the brain (occipital lobe).

Visual Tract.—The visual tract, or visual conduction paths, may briefly be described as follows:

The retina is a highly developed structure, which, from the histologic standpoint, may be divided into three layers: (1) The *layer of the neuro-epithelium*, composed of two strata, namely, the *layer of rods and cones* and the *external nuclear layer*, the former constituting the specialized outer portions and the latter the nucleated bodies of the *visual cells;* (2) the *layer of the bipolar cells*, which by some authorities are looked upon as the peripheral visual neurons; (3) the *layer of the ganglion-cells.*

The long processes, or axons, of the ganglion-cells pass into the *nerve-fiber layer of the retina*, reaching the *papilla* or *nerve-head*, and proceed to the *optic nerve.* Having reached the *optic chiasm*, a portion of the fibers of one optic nerve cross over and enter the *optic tract* of the opposite side, forming the *crossed fasciculus*, while a certain number of other fibers do not cross, but enter the optic tract of the same side, forming the *non-crossed fasciculus.* The non-crossed fasciculus arises chiefly from the temporal side of the retina, while the crossed fasciculus arises from the ganglion-cells of the nasal side of the retina. The bundle from the macula lutea, called the *macular fasciculus*, or *papillomacular bundle*, in general terms, is situated in the central part of the optic nerve and maintains its central position in the optic chiasm and in the optic tract, and is composed of crossing and direct fibers. The optic tract on each side behind the chiasm passes around the cerebral peduncle of the same side and arrives at the junction of the interbrain and midbrain, and divides into a lateral and a medial root. The fibers of the lateral root terminate in the *lateral geniculate body*, in the *pulvinar* of the *thalamus*, and in the *superior colliculus of the corpora quadrigemina.* These structures have been designated the *primary visual ganglia* or *primary optic centers.*[2] The corpora quadri-

[1] The terms *hemiopia* and *hemianopsia* are often used synonymously. Really, hemiopia signifies loss in the perceptive power of one-half of the retina, while hemianopsia means obscuration or loss of one-half of the visual field (Seguin). Other names which are used are *hemianopia* and *hemiablepsia.*

[2] According to W. G. Spiller, the chief "primary" optic center is the external geniculate body. The pulvinar of the optic thalamus is also an important "pri-

gemina are not concerned in the act of vision, but in the activity of the pupil. The medial root of the optic tract has no connection either with the retina or with the optic centers of the interbrain and midbrain.

From the regions just described fibers proceed backward through the posterior part of the *internal capsule* to the cortex. These are the *optic* or *visual radiations*, or *fibers of Gratiolet* or *of Wernicke*. Passing through the internal capsule, they cross the sensitive fibers coming down from the hemisphere, and spreading out like a fan, rise upward, wind outside of the tip of the lateral ventricle, to reach their

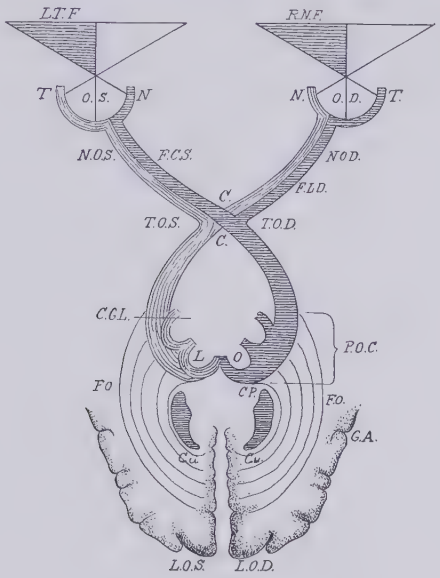


FIG. 258.—Diagram illustrating the visual path and its relation to the visual field, left lateral hemianopsia being shown (Seguin): *L. T. F.*, left temporal half-field; *R. N. F.*, right nasal half-field; *O. S.*, left eye; *O. D.*, right eye; *N.*, nasal, and *T.*, temporal halves of the retinas; *N. O. S.*, left optic nerve; *N. O. D.*, right optic nerve; *F. C. S.*, left crossed fasciculus; *F. L. D.*, right lateral or non-crossed fasciculus; *C.*, chiasm or decussation of the fasciculi; *T. O. D.*, right optic tract; *T. O. S.*, left optic tract; *C. G. L.*, corpus geniculatum laterale (medial corpus geniculatum and its arms are omitted); *L. O.*, optic lobes (corpora quadrigemina); *P. O. C.*, primary optic centers (including corpora quadrigemina, corpora geniculata, and pulvinar of the optic thalamus); *F. O.*, optic fasciculus, radiating visual fibers of Gratiolet in the internal capsule; *C. P.*, posterior horn of the lateral ventricle; *G. A.*, region of the gyrus angularis; *L. O. S.*, left occipital lobe; *L. O. D*,. right occipital lobe; *Cu.*, cuneus and subjacent gyri constituting the cortical visual center in man. The shaded lines represent the parts connected with the right halves of the retinas.

destination at the lower part of the median surface of the *occipital lobe*—that is, the cortical termination of the visual tracts. The exact area occupied by the cortical center of vision has not been determined. In general terms it is situated about the *cuneus* and *calcarine fissure*, and does not comprise the whole of the occipital lobe.

mary" optic center. The anterior colliculus of the quadrigeminal body in man has an unimportant relation to vision. The hypothalamic body, the habenula, and the internal geniculate body probably are not part of the visual system (consult Spiller, "A Case of Complete Absence of the Visual System in an Adult," Univ. of Penna. Medical Bulletin, February, 1902).

By comparing the description of the varieties of hemianopsia which follow with Fig. 258 the student will understand the mechanism of their development.

Varieties of Hemianopsia.—Hemianopsia is divided into *horizontal*, in which the dividing line between the darkened and preserved field is horizontal; and *vertical*, in which the dividing line is vertical.

1. *Horizontal hemianopsia* (*altitudinal*) may be inferior or superior, both lower or both upper half-fields being wanting. In addition to diseases of the eye, it is possible that such a condition can arise under the influence of a lesion so situated as to press upon the upper or lower part of the chiasm, or downward upon one optic tract, or upon the lower or upper part of both optic nerves. A double lesion in front of the chiasm may produce loss of the upper half of the field in one eye and of the lower half of the field in the other eye.

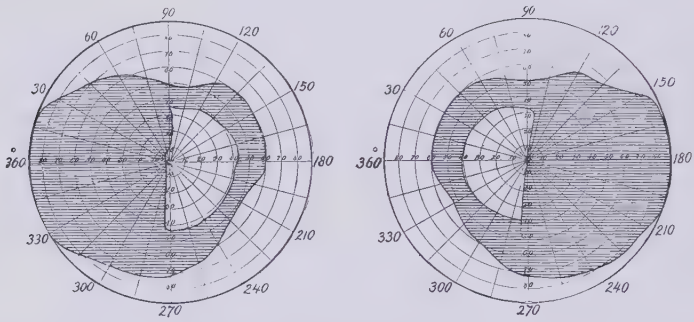


FIG. 259.—Bitemporal hemianopsia. The shaded areas represent the portions of the fields which are dark, and it is evident that there are entire loss of both temporal fields and some contraction of the preserved fields (from a case of acromegaly).

2. *Vertical Hemianopsia.*—This is subdivided into several varieties:

(*a*) *Bitemporal hemianopsia* (peripheral), in which both temporal fields are wanting, is characteristic of lesion of the chiasm. The defect is not necessarily complete from the beginning. Color-sense at first may be alone affected, followed later by loss of form- and light-sense. In place of complete bitemporal hemianopsia there may be paracentral or bitemporal hemiopic scotomas, which gradually broaden into bitemporal hemianopsia (see also pages 589 and 590). There are a number of types or "stages" of bitemporal visual field defects. Wildbrand described nine. Cushing and Walker have elaborated his list.

(*b*) *Binasal hemianopsia*, in which both nasal fields are wanting, is comparatively rare. Unilateral nasal hemianopsia also occurs.

(*c*) *Homonymous hemianopsia* (central), in which the corresponding half of the field of each eye is wanting: thus, both right or both left fields may be darkened, in the former case indicating loss of function of the left half of each retina and designated *right homonymous lateral hemianopsia*, and in the latter case indicating loss of function of the right half of each retina, and designated *left homonymous lateral hemianopsia* (see Figs. 260 and 261).

Hemianopsia may be *complete*—*i. e.*, the entire half of each field is wanting—or *incomplete*, *i. e.*, a portion of each half-field is wanting, the defect usually being in the form of a quadrant (Figs. 260–262). The preserved half-fields may be of their normal size, or they may exhibit concentric contraction. Asymmetry of the fields in homonymous hemianopsia has often been observed. The field homolateral to the lesion

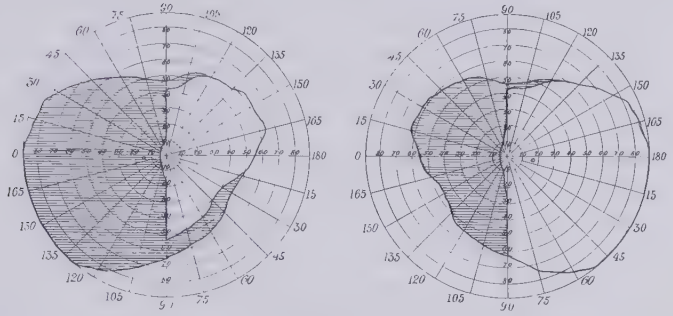


FIG. 260.—Left homonymous hemianopsia, from a case of gunshot wound, with suspected lesion of the right cuneus. The shading shows where vision was lost (from a patient under the care of the late Dr. S. Weir Mitchell in the Infirmary for Nervous Diseases).

may be more defective than the contralateral field in tumors of the temporal lobe (Cushing). Sometimes a hemianopic defect exists in one field and quadrantic defect in the other, or one field may be hemianopic and the other concentrically contracted.

Finally, the hemianopsia may be *absolute*—*i. e.*, all the three functions of sight (perception of light, of form, and of color) are wanting—

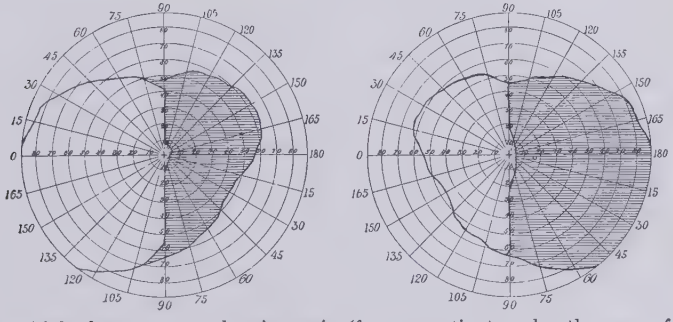


FIG. 261.—Right homonymous hemianopsia (from a patient under the care of the late Dr. Wharton Sinkler).

or it may be *relative*, *i. e.*, perception of color only is lacking, light-sense and form-sense being preserved; or perception of color and form is wanting in the deficient area of the field, but the light-sense is preserved. Those cases in which the half-defect is present for colors alone are described under the name *homonymous hemiachromatopsia*. They have been attributed to a cortical lesion of less intensity than one which produces absolute hemianopsia. In a remarkable case of this kind which

the author studied with Dr. J. William White, at the onset the hemianopsia was absolute; later light-sense and form-sense returned. The obliteration of the color-sense remained, although in all other respects the patient recovered. Sir Gordon Holmes, however, doubts if it has been conclusively shown that color perception may be lost in any part of the field when that of light or white is undisturbed; in short, his observations tend to show that an isolated loss or dissociation of color vision is not produced by cerebral lesions.

The dividing line in hemianopsia may exactly cut the fixing-point, or it may pass around this point and leave it within the region of preserved vision. When the separation between the halves of the field of vision does not lie in the vertical meridian, but, on account of differences in anatomic conditions is situated more or less beyond it, it is called *overshot field of vision* (Wilbrand). The want of uniformity between the seeing and the blind areas may be manifested by the failure of the dividing line to coincide with the vertical meridian for some distance, by its assuming an oblique or irregular direction, or by forming

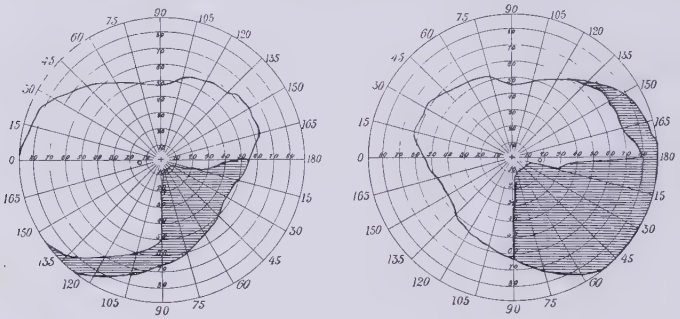


FIG. 262.—Quadrant homonymous anopsia or tetranopsia, shading as before. A quadrant of each field is wanting. The lesion was probably in the cuneus.

an open angle. The borderline between the blind and seeing halves of the field is more irregular in lesions of the chiasm than in those posterior to this position.

A number of cases of *double* homonymous hemianopsia have been recorded due to a cerebral lesion on each side of the brain. In these circumstances there is usually preserved a small central field of each eye, that is, the macula is exempted. Small homonymous paracentral scotomas, the so-called *macular hemianopsia*, have been reported (Wilbrand). Central vision may be good, but certain visual acts, for instance, reading, are much disturbed.

Significance of Hemianopsia.—Typical bitemporal hemianopsia of permanent character is caused by a lesion—tumor, pituitary body disease, aneurysm, exostosis, arterial disease, basal syphilis, tuberculosis, etc.—which destroys the conductivity of both crossed fasciculi, leaving the non-crossed fasciculi unaffected (see Fig. 258). It is a common symptom of pituitary body disease (neoplasm or struma). Affections of this region, however, may cause other varieties of visual field

defects, especially paracentral scotomas as noted on page 590, and the hemianopsia caused by pituitary body lesions is not always bitemporal.

A true chiasmal variety of binasal hemianopsia probably does not occur, although the visual field defect has been attributed to disease of the lateral angles of the chiasm. It may be due to vascular pressure exerted on the optic nerves on account of sclerosis of the vessels. Binasal hemianopsia in old people may be due to this cause (E. Fuchs). The terminal stage of visual field deformation in choked disk may take the form of a binasal hemianopsia (Cushing and Walker). Some of the cases seem best explained by a bilateral inflammation of the trunks of the optic nerves in front of the chiasm. Unilateral hemianopsia, if not caused by disease within the eye, could originate from an injury or from a lesion in one optic nerve. A nasal hemianopsia on one side could be produced by a lesion affecting the lateral portion of the chiasm involving the non-crossing fibers of one eye.

Homonymous lateral hemianopsia is caused by a lesion situated in the occipital lobe, the optic radiations, the internal capsule, the primary optic centers, or the optic tract (see Fig. 258).

(*a*) The lesion in hemianopsia is on the opposite side of the dark fields.

(*b*) If the preserved fields are accompanied by concentric contraction, the smaller half-field will be in the eye opposite to the lesion (Seguin, see also p. 295); contraction of the preserved half-field is more common with lesions of the cortex, but also may occur in lesions of the tractus.

(*c*) If the hemianopsia is relative, the lesion is probably in the cortex, but cortical lesions are not excluded by absolute hemianopsia.

(*d*) A lesion confined to the cuneus, or to it and the gray matter immediately surrounding it, on the mesial surface of the occipital lobe, produces homonymous lateral hemianopsia without motor or sensory symptoms, at least without these as a direct consequence of the lesion, although they may appear as *indirect*, or, as they are sometimes called, *distant symptoms*.

(*e*) A lesion producing typical hemiplegia, aphasia, if the right side is paralyzed, little or no anesthesia and lateral hemianopsia, is probably due to disease in the area supplied by the middle cerebral artery.

(*f*) A lesion causing hemiplegia, hemianesthesia, and lateral hemianopsia is probably situated in the posterior portion of the internal capsule.

(*g*) A lesion causing hemianesthesia, ataxic movements of one-half of the body, no distinct hemiplegia, and lateral hemianopsia could be situated in the posterior lateral part of the optic thalamus.

(*h*) A lesion causing the symptoms of disease of the base of the brain, associated at the same time with changes in the pupil, changes in the nerve-head, and lateral hemianopsia, could be situated in one optic tract or in the primary optic centers on one side.

(*i*) Incomplete hemianopsia, assuming usually a quadrant-shaped defect, may be present on account of a lesion confined to the lower half of the cuneus. It may also occur with less definite limitations in lesions of the subcortical substance of the occipital lobe and then may be associated with other symptoms, as hemiplegia and hemianesthesia. It may be due to a lesion of the tract, but then will be accompanied by other symptoms indicating basal disease, or to a lesion of the external geniculate body. *Quadrantic hemianopsias* may result from lesions of the optic radiations (Gordon Holmes). (See also page 596.)

(*j*) A hemianopsia in which there is preservation of the light-sense, but loss of either the color-sense or the form-sense, has been attributed to a lesion in the cortex of the visual center. (See also page 596.)

During the recent war the opportunities of studying the effect of injuries of the brain on the visual field were extensive, and a large literature on this subject is available which could not be analyzed in a book of the present scope. The conclusions reached by Sir Gordon Holmes and Sir William Lister on the localization and organization of the cortical centers of vision and the visual disturbances which may be produced by lesions in different portions of the brain, differ in some respects from those which have been previously held, for example, those of Henschen. They are as follows:

"1. The upper half of each retina is represented in the dorsal, and the lower in the ventral, part of each visual area.

"2. The center for macular or central vision lies in the most posterior part of the visual cortex, probably on the margins and on the lateral surfaces of the occipital poles. The macula has not a bilateral representation.

"3. The center for vision subserved by the periphery of the retinæ is situated in the anterior portions of the visual areas, and the serial concentric zones of the retinæ from the macula to the periphery are probably represented in this order from behind forward in the visual cortex.

"4. Those portions of the retinæ adjoining their vertical axes are probably represented in the dorsal and ventral margins of the visual areas, while the retina in the neighborhood of its horizontal axis is projected on to the walls and the floor of the calcarine fissures.

"5. Severe lesions of the visual cortex produce complete blindness in the corresponding portions of the visual fields, or if incomplete, an amblyopia, color vision being generally lost and white objects appearing indistinct, or only more potent stimuli, as abruptly moving objects, may excite sensations.

"6. The defects of vision in the fields of the two eyes are always congruous and superimposable, provided that no abnormality of the peripheral visual apparatus exists.

"7. Lesions of the lateral surface of the hemispheres, particularly of the posterior parietal regions, may cause certain disturbances of the higher visual perceptual functions with intact visual sensibility, as loss of visual orientation and localization in space, disturbance of the perception of depth and distance, visual attention loss, and visual agnosia."

George Riddoch, as the result of visual field studies in cases of occipital injury, has demonstrated that should recovery of vision occur in restricted fields of vision the first visual stimulus perceived, capable of being recorded on a chart as a field, is *movement*, which begins in the peripheral field; the field for the appreciation of movement, being the more primitive perception, is larger than the one obtained by the recognition of a test-object. His observations also agree with the conclusions reached by Holmes and Lister in the cortical representation of the retina.

The Pupil in Hemianopsia.—The reaction of the pupil in hemianopsia has usually been regarded as a localizing symptom of importance; this is denied by some observers (see foot note, page 599). Generally the rules are stated as follows:

If, in hemianopsia, the light thrown upon either the blind or the seeing side of the retina causes contraction of the pupil, the lesion is back of the primary optic centers.

If there is no contraction of the pupil when the ray of light falls upon the blind side of the retina, but there is contraction when it falls upon the seeing side, the lesion is in front of the primary optic centers.

In the former instance the lesion is so situated that there is no dis-

turbance of the sensorimotor arc of the pupils; in the latter the lesion interferes with this arc, and the phenomenon receives the name *hemiopic* or *hemianopic pupillary inaction.* It is often called *Wernicke's symptom.*[1]

[1] Henschen (Klin. med. anat. Beiträge zur Pathologie des Gehirns, Th. iii) concludes that the hemiopic pupillary inaction (abbreviated H. R.) is present in tract lesions, even when minute or merely caused by pressure; lesions of the posterior segment of the thalamus and pulvinar—perhaps from pressure on the tract, or by destroying the brachium anterius; lesions of the chiasm (occasionally absent for unknown reasons); and in lesions of the nerve, with unilateral hemianopsia. It is a difficult symptom to demonstrate (Henschen uses a special lamp) and its existence is doubted by some observers. The iris reaction may not be entirely absent when the ray falls on the blind side of the retina, but it is much less marked than the one which follows light stimulus of the opposite side. C. B. Walker (Trans. Section on Ophthalmology, Amer. Med. Assoc., 1914) has designed a clinical instrument for elucidating the hemiopic pupillary reaction and concludes from his studies that it has no topical diagnostic value. To replace Henschen's phenomenon, Wilbrand has proposed a prism test. It was thus described by Saenger: The patient fixes a white point on a large black plane surface. Suddenly two prisms of equal degree (15°) are brought before both eyes, their apices being turned to the hemianopic defect. If cortical hemianopsia exists, the patient's eyes will move so that the fovea is directed to the object; if the reflex path is interrupted by a lesion of the tractus, there is no movement of the eyes.

CHAPTER XVIII

MOVEMENTS OF THE EYEBALLS AND THEIR ANOMALIES

Anatomy and Physiologic Action of the Ocular Muscles.—The movements of the eye are controlled by the action of six muscles, four straight and two oblique, in general terms situated in the orbital region.

1. The *external rectus* arises by two heads, respectively from the outer margin of the optic foramen and the common tendon of the inferior and internal recti, and in part from a process of bone on the lower margin of the sphenoid fissure. Its tendon is inserted into the sclera 7 mm. from the margin of the cornea. It is supplied by the *sixth* or *abducens nerve*. Its pre-eminent[1] action is *abduction*—that is, it rotates the eye directly outward.

2. The *internal rectus* arises from the optic foramen by a tendon common to it and the inferior rectus, and passes forward to be inserted by a tendinous expansion into the sclerotic coat 5 mm. from the margin of the cornea. It is supplied by one of the three branches of the inferior division of the *third* or *oculomotor nerve*. Its pre-eminent action is *adduction*—that is, it rotates the eye directly inward.

3. The *superior rectus* arises from the upper margin of the optic foramen and from the fibrous sheath of the optic nerve, and is inserted by a tendinous expansion into the sclerotic coat 8 mm. from the margin of the cornea. It is supplied by the superior division of the *third* or *oculomotor nerve*. Its pre-eminent action is *elevation* or *superduction*—that is, it rotates the eye upward. It also adducts it and rotates the upper end of the vertical meridian of the cornea inward (*inward torsion* or *intorsion*).

4. The *inferior rectus* arises from the optic foramen by a tendon common to it and the internal rectus and passes forward to be inserted by a tendinous expansion into the sclerotic coat, 6 mm. from the margin of the cornea. It is supplied by one of the three branches of the inferior division of the *third* or *oculomotor nerve*. Its pre-eminent action is *depression*, or *subduction*—that is, it rotates the eye downward. It also adducts it and rotates the vertical meridian of the cornea outward (*outward torsion, extorsion*).

5. The *superior oblique* (trochlear) is situated at the upper and inner side of the orbit, and arises above the inner margin of the optic foramen. It proceeds to the inner angle of the orbit, at which point its rounded tendon passes through a fibrocartilaginous pulley occupying a fossa just within the supra-orbital margin of the frontal bone, and is next reflected backward, outward, and downward, to be inserted about 18

[1] This term is borrowed from Maddox.

mm. from the edge of the cornea between the superior and external recti. It is supplied by the *fourth* or *trochlear nerve*. Its pre-eminent action is *intorsion*—that is, it rotates the vertical meridian inward. It also depresses the eye and abducts it (see footnote).

6. The *inferior oblique* is situated at the bottom of the orbit and arises from a depression in the orbital plate of the superior maxillary bone. Passing beneath the inferior rectus, it is directed outward, backward, and upward, and reaches its insertion into the sclera by means of a thin tendon about 19 mm. from the corneal margin, within the position of the external rectus. It is supplied by the largest branch of the superior division of the *third* or *oculomotor nerve*. Its pre-eminent action is *extorsion*—that is, it rotates the vertical meridian outward. It also elevates the eye and abducts it.[1]

The starting-point from which the actions of the muscles are reckoned is the *primary position* of the globe, defined by Mauthner as that position of the eyes from which the visual lines can be moved without the eyes being revolved around their anteroposterior axes. The eyes occupy about this position when they are directed straight forward, the head being held erect, and a distant object, situated in the median line of the visual plane, is observed with practically parallel visual lines. Positions of the eyes other than this are called *secondary positions*.

Rotation of the Eyeball Around the Visual Line.—If a *vertical line* is passed through the *visual line* so as to divide the eyeball into two lateral halves, it will intersect the surface of the eyeball in what is called the *vertical meridian*. The latter may be defined with sufficient accuracy as a line passing through the center of the pupil in a direction perpendicular to the line joining the centers of the two pupils. It joins the uppermost and lowermost points of the corneal margin.

In movements of the eyeball directly upward (combined action of the superior rectus and inferior oblique) or downward (combined action of the inferior rectus and superior oblique), or directly inward or outward, the vertical meridian remains vertical.

In *oblique* movements of the eyeball, upward and inward (superior and internal rectus, with inferior oblique); downward and inward (inferior and internal rectus, with superior oblique); upward and outward (superior and external rectus, with inferior oblique); or downward and outward (inferior and external rectus, with superior oblique), the vertical meridian will be observed to rotate like the spokes of a wheel (*wheel-rotation* or *torsion*). The eyeball appears to rotate around the visual line; this is effected by the superior and inferior recti and the superior and inferior oblique muscles. The upper extremity of the vertical meridian of the cornea is deviated outward (toward the temple) by the inferior recti and inferior oblique muscles; and inward (toward the nose) by the superior recti and superior oblique muscles. The

[1] Duane, basing his opinion on the results of paralysis, believes that *depression* is the most important action of the superior oblique and that *elevation* is the most important action of the inferior oblique, intorsion and extorsion, respectively, being subsidiary actions.

deviation of the vertical meridian produced by any muscle is greatest when the axis of rotation for that muscle coincides with the visual line.

The superior and inferior recti exercise the greatest degree of torsion when the eyeball is drawn toward the nose, and either upward or downward.

The oblique muscles, on the contrary, exercise their maximum amount of torsion when the eyeball is drawn toward the temple, and either upward or downward. The inferior oblique, while it aids the superior rectus in upward movements, antagonizes it in the rotation of the vertical meridian and the movement of the eyeball inward.

The visual line coincides most nearly with the axis of rotation of the superior and inferior recti when the eyeball is drawn toward the nose, and most nearly with that of the superior and inferior oblique muscles when the eyeball is turned toward the temple. The superior oblique aids the inferior rectus in drawing the eye downward, but antagonizes it in the rotation of the vertical meridian and in the movement of the eyeball inward.

In extreme diagonal movements of the eyes the action of the obliques and the recti is to make the vertical meridian tilt toward the nose, or temple. But if the muscles are evenly balanced, the vertical meridians of the two eyes, however tilted, remain parallel, and under these conditions the retinal images are projected normally, vertical objects still appearing vertical. According to Sherrington our notions of the verticality of the objects are dependent not only on visual but on other sensory impressions.

In paralysis of the eye muscles the vertical meridians no longer remain parallel, and the image of one eye appears oblique with respect to the other. (See also page 607.)

Associated Movements.—Except in pathologic circumstances, there is coördination in the movements of the eyes, and the movement of one eyeball is associated with that of its fellow. In other words, both eyes are used for seeing (*binocular vision*), and are so adjusted that the image of the object regarded falls simultaneously on both maculæ (*binocular fixation*). If a distant object is to be looked at and the right eye is turned to the right, the left eye is also turned to the right and to the same extent as its fellow, because of the associated action of the external rectus of the right eye and the internal rectus of the left eye under the same innervation impulse. If one eye is elevated, the other is also elevated; if one is depressed, the other is also depressed. These are associated movements in the same direction.

If a near object is to be looked at, the visual axes converge for the point at which it is situated because of the associated action of the internal recti of the two eyes (*convergence* or *accommodative* movement); if the eyes are removed from this point and directed to a distant object, the visual axes tend to parallelism because of the action of both external recti.

If the associated movements of the eyes were not thus regulated by equal impulses from the coördinating center, single vision would not

be possible, because the images of any object would not fall upon *corresponding points* of the two retinas. Inasmuch as every normal individual has two normally constructed eyes, he must receive from every object two sets of sensations, which are blended into one when the movements of the eyes are so arranged that the images fall upon corresponding retinal areas.

A point situated anywhere upon the right side of one retina has its corresponding point upon the right side of the other retina, and points on the left side of one correspond with points on the left side of the other. The upper half of the retina of the right eye corresponds to the upper half of the retina of the left eye, and the lower half of the right to the lower half of the left; the nasal side of the right eye corresponds with the malar side of the left, and the malar of the right with the nasal of the left. If, for any reason, the movements of the eyes become disarranged so that the images do not fall upon corresponding or identical retinal areas, the images become double.

The desire for *binocular single vision*, or single vision with the two eyes, which depends upon the blending of the two sets of sensations, or, as it is also called, *fusion*, is believed to be the origin of the impulse which directs the movements of the eyeballs, especially in association in the same direction.

In addition to this desire for blending the two sets of sensations into one, in the associated movements of the eyes in the same direction, there is also another regulating factor—*i. e.*, the connection between convergence and accommodation (see page 44).

Overcoming Prisms.—When a prism is placed before one eye with its base inward and diplopia is produced, an outward rotation of the eye occurs, and when the prism is placed with its base outward, an inward rotation of the eye takes place, and the influence of the prism is overcome, so that single vision again is possible within the limitations which have been recorded on page 78, where *prism-convergence*, *prism-divergence*, and *sursumvergence* are described.

Field of Fixation.—This includes all points which the eye under observation can successively fixate, the head being perfectly stationary. The field of fixation of an amblyopic eye may be determined by watching the image of a candle-flame on the center of the cornea as the eye follows the test-light moved along the perimeter arc until the limit of movement is reached. Ordinarily the patient should be seated in the position for testing the visual field before the perimeter, with the semicircle horizontal, and the eye (the head being rigid) made to follow a word composed of small test-letters, and the point where vision ceases to be distinct marked on successive meridians. In place of letters, two fine dots set close together on a card may be employed, and the point noted where the dots cease to appear as two.

Landolt's measurements of the field of fixation under normal conditions are as follows: Outward, 45–50; inward, 45; upward, 35–40; downward, 60. Duane's average measurements are: Outward, 51; inward, 53; upward, 43; downward, 63.

G. T. Stevens determined the rotations of the eyes with a special instrument called a *tropometer* (page 802). According to him, the most favorable rotations are: Outward, 50; inward, 50; upward, 33; downward, 50.

Strabismus, Squint, or Heterotropia.—Under the general term *strabismus* or *squint* are included those conditions which occur when the visual axis of one eye is deviated from the point of fixation. The

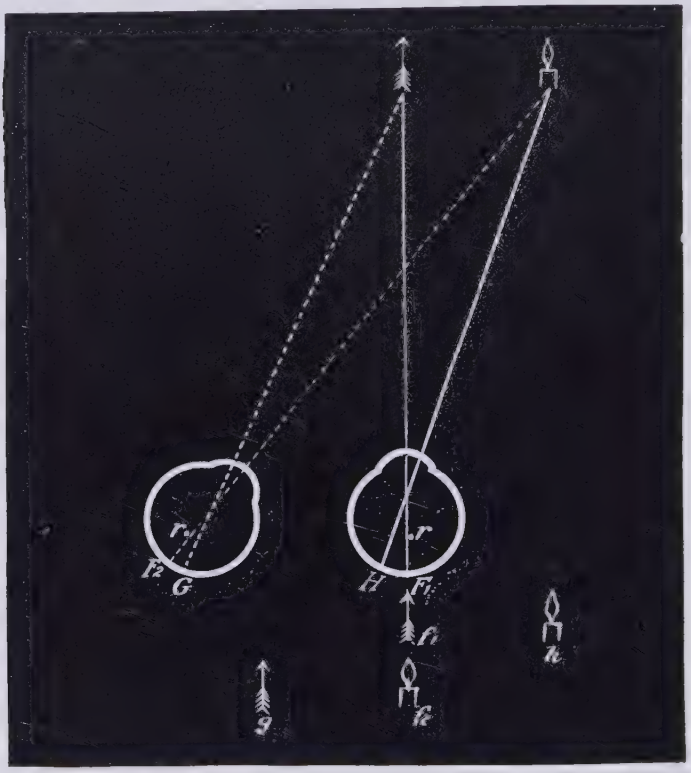


FIG. 263.—Convergent strabismus. Position and projection of the images (James Wallace).

This diagram also illustrates the principle of *regional exclusion.* If in the case pictured there is squint with suppression, then according to this principle as enuciated by von Graefe, not only is the arrow (the image of which falls on the right macula, F_1) not seen by the left eye, but the candle (the image of which falls on the macula of the left eye) is not perceived by the right eye.

eye the visual axis of which is directed to the object fixated is termed the *fixing* (*fixating*) *eye;* the other eye is termed the *squinting* or *deviating eye.* The deviation may be inward (*strabismus convergens*), outward (*strabismus divergens*), upward (*strabismus sursum vergens*), or downward (*strabismus deorsum vergens*).

1. **Convergent Strabismus, or Esotropia.**—In this form of squint the visual line of one eye is directed to the object fixed. The visual line

of the other eye is deviated inward, and intersects that of the sound eye at some point nearer than the object fixated. The image of an object situated on the visual line of this eye would be formed on the fovea, and projected to the same point in the field of fixation.

Figure 263 represents a convergent squint of the left eye, and serves to explain the results of an inward deviation of one eye from any cause.

The center of rotation is seen at r. The arrow is the object fixated; its image is formed on the fovea of the right eye, F_1, and its position in the field is denoted by f_1. The candle forms its image on the retina of the right eye to the left of the fovea at H; its image is properly projected to the right, and its position in the field is denoted by h. The visual axis of the left eye is directed to the candle; its image is formed on the fovea at F_2, and its position in the field is denoted by f_2, identical with that of f_1, because formed on an identical point of the retina. The arrow forms an image on the retina of the left eye at G, to the right of the fovea; it is consequently projected to the left of that of F_2, and its position in the field is denoted by g.

The right eye projects images correctly; the left eye projects them to the left of their true position—*i. e.*, to the side of the squinting eye. The diplopia is *simple* or *homonymous*.

2. **Divergent Strabismus, or Exotropia.**—In this form of squint the visual line of one eye fixates the object, while the visual line of the other eye lacks the necessary movement inward to intersect that of its fellow at the point of fixation.

As long as the visual axis of the affected eye intersects that of the sound eye in its anterior extremity, the condition may be denominated *insufficiency of convergence*. When the visual axes no longer intersect anteriorly, but diverge from each other so that their posterior extremities intersect, the condition may be denominated *divergent squint*. If the convergence insufficiency is considerable, so that the visual axes do not intersect at the ordinary reading distance, although they do intersect anteriorly, the condition is denominated by many as a *directly periodic divergent squint*. On the other hand, there are not a few cases in which the axes diverge at their anterior extremities but intersect at the reading distance. These may be denominated cases of *inversely periodic divergent squint*, being, in fact, due to a condition of marked divergence excess (Duane).

Figure 264 represents a divergent squint of the left eye, and explains the effects of an outward deviation of one eye from any cause upon the position of the image of an object which is fixated.

The center of rotation is at r. The arrow is the object fixed; its image is formed on the fovea of the right eye at F_1, and its position in the field is denoted by f_1. The candle forms its image on the retina of the right eye to the right of the fovea at H; its image is properly projected to the left and its position in the field is denoted by h. The visual axis of the left eye is directed to the candle; its image is formed on the fovea at F_2, and its position in the field denoted by f_2, identical with that of f_1, because formed on identical points of the retina. The arrow forms an image on the retina of the left eye at G, to the left of the fovea; it is consequently projected to the right of that of F_2, and its position in the field is denoted by g.

The right eye projects images properly: the left eye projects them to the right of their true position—*i. e.*, the side opposite to the squinting eye. The diplopia is *crossed* or *heteronymous*.

3. **Upward and Downward Squint, or Hypertropia.**—If vertical deviation (upward or downward) causes diplopia, the images are on different levels, the upper image corresponding with the lower eye. Simple vertical deviation without lateral is rare. Generally in lateral strabismus the squinting eye deviates upward, but may also turn downward (Schweigger). According to Hansell, functional internal squint (*esotropia*) is always associated with upward deviation (*hyper-*

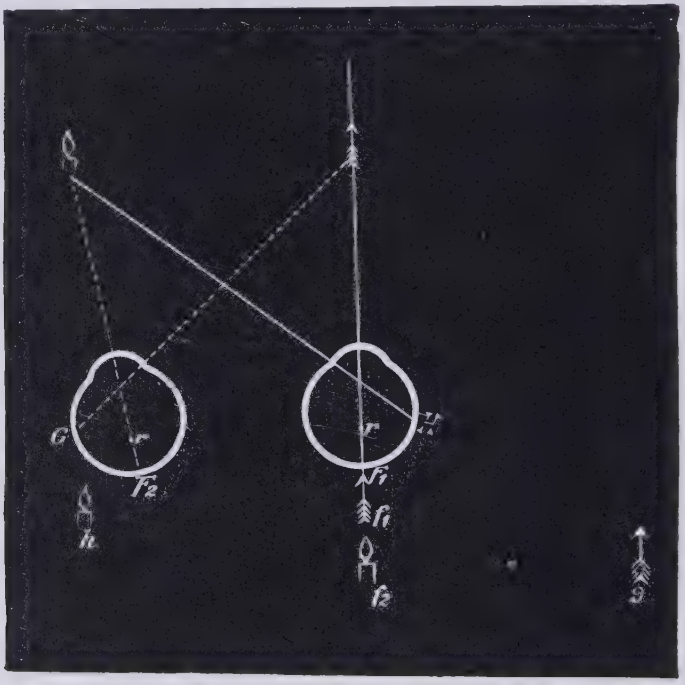


FIG. 264.—Divergent strabismus. Position and projection of the images (James Wallace).

tropia). In deviations, especially when there is vertical squint, one of the images is often oblique with respect to the other.

This obliquity can be simplified for study by dividing it into two kinds: either the vertical meridians incline toward each other by their upper extremities, or else they diverge from each other.

The meridians *diverge* from each other when the upper extremity of one vertical meridian is directed toward the temple, while the vertical meridian of the other eye remains perpendicular. If the two eyes are sighting an upright object, like a candle, the latter will form a vertical inverted image on the retina of each. In the eye whose vertical meridian remains perpendicular, this image will coincide with that

meridian. In the eye whose meridian is tilted toward the temple, the image will fail to coincide with the vertical meridian, the lower image of the candle flame lying below the macula and somewhere on the temporal half of the retina, and the image of the candle base lying upon the macula and somewhere on the nasal half of the retina. In accordance with the law of projection, images on the nasal half of the retina are referred to the temporal portion of the field, and images on the temporal half of the retina are referred to the nasal portion of the field. With the vertical meridian tilted toward the temple the

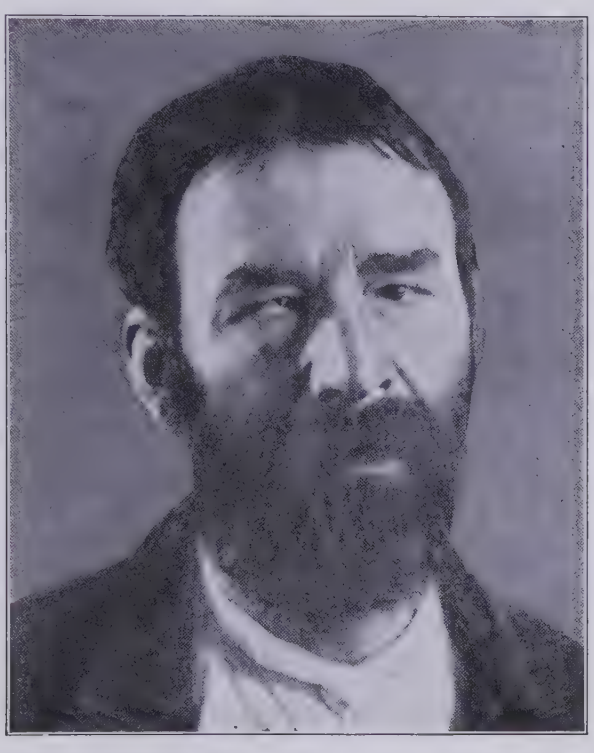


FIG. 265.—Convergent strabismus with decided upward deviation (from a patient in the Philadelphia General Hospital).

candle forms an image on the retina which is projected outward, so that it seems to converge by its upper extremity toward that of the other eye when the diplopia is homonymous; when crossed diplopia exists, it seems to diverge.

The meridians *converge* toward each other when the upper extremity of one vertical meridian is tilted toward the nose, while the vertical meridian of the other eye remains perpendicular.

When the vertical meridian is tilted toward the nose by its upper extremity, the image of the candle occupies, with its lower portion, a point in the nasal half of the retina, and with its upper portion a point

in the temporal half of the retina. It is projected outward in such a manner that it seems to lean away from the image of the other eye when the diplopia is homonymous; when crossed diplopia exists, it seems to lean toward the image of the other eye.

In paralysis of the ocular muscles it is usually the image of the paralytic eye which appears oblique. Sometimes, however, the patient regards this image as vertical and the image of the sound eye as oblique (see also pages 601 and 602).

Paralysis of the Exterior Ocular Muscles (*Paralytic Strabismus*). —This may be *complete* (the muscle is entirely paralyzed) or *incomplete* (the muscle is partially paralyzed).

A. **General Symptoms.**—Certain symptoms are common to paralysis of the exterior eye muscles.

1. *Loss of Binocular Single Vision, or Diplopia.*—The cause of this, evident from the previous explanations, depends upon the deviation of the affected eye so that the images from an object are no longer fused, owing to their failure to fall upon "identical points" in the two retinas. Diplopia increases as the object is moved to the side of the paralyzed muscle. In slight degree it amounts only to indistinct vision.

2. *Non-correspondence of the Direction of the Two Eyes, or Strabismus.*—This depends upon the deviation to which the affected eye is subjected by the tone of the unresisted action of the muscle which is the antagonist of the paralyzed muscle, and also, in part, upon the effect of secondary contractures. Squint is not always plainly manifest, and may appear only if an attempt is made to move the eye in the direction of the action of the palsied muscle.

3. *Loss or Limitation of Movement* (*Primary Deviation*).—The limitation of movement is always in the direction of the action of the affected muscle; consequently the deviation of the eye is in a direction opposite to the action of the muscle.

4. *Deviation of the Sound Eye, While the Affected Eye Fixates* (*Secondary Deviation*).—During the act of fixation by the affected eye the same degree of nervous impulse passes from the center to the muscles of the affected eye and to those of its non-affected associate; the former requires an abnormally great impulse to stimulate its movement, and hence the latter is overexcited, and the resulting movement is excessive. The secondary deviation, therefore, is greater than the primary deviation.

In order to demonstrate this the sound eye is covered with the hand, while the affected eye is directed toward an object held at a distance of about 1 foot. The covering hand is then moved from the sound to the affected eye. In order to fixate the object, the sound eye must now move in a direction opposite to that toward which the paralyzed muscle rotates the ball. This backward movement represents the degree of previous excess called into existence by the undue amount of nerve force which the normal muscle originally received. Thus primary and secondary deviations are in opposite direction, but both in the line of action of the affected muscle.

5. *False Projection of the Field of Vision.*—This depends upon an inaccurate estimation of the position of an object situated in such a portion of the visual field that it requires an effort on the part of the affected muscle to turn the eye toward it. A normal individual (his head being stationary, and one eye being closed, *e. g.*, the right) can readily and accurately touch an object lying within his reach to the left of the median line, because the degree of innervation required to make the lateral movement of the eye in order to see the object gives the necessary information, based on experience, how far to the left the object lies. In the same circumstances an individual with a paretic left external rectus, instead of touching the object, would pass his hand beyond it—*i. e.*, to the left of it, because the excessive innervation which is now necessary to make the lateral turn gives the impression that the object lies farther to the left. In other words, the object is projected to a position in the visual field which it does not have.

6. *Vertigo.*—This depends, both eyes being open, upon the diplopia and the confusion arising from trying to distinguish between the real and the false image. If one eye (the unaffected eye) is closed, it depends upon the condition described in the preceding paragraph.

In a paretic condition of the muscles which rotate the eye downward vertigo may result from an erroneous localization of objects in the lower field, as they seem to lie in a plane deeper than they really are.

Patients with ocular palsies commonly close the affected eye, although closure of either eye would remove the diplopia.

7. *Altered Position of the Carriage of the Head.*—This depends upon the impulse of the patient to carry his head in that direction in which he is least troubled by the double images, and this is usually in the direction toward which the affected muscle moves the eye. In vertical deviation the head is often tilted toward one shoulder—toward the side of the higher eye if the hyperphoria or hypertropia is combined with crossed diplopia, and toward the other side if the hyperphoria or hypertropia is combined with homonymous diplopia.

B. **Varieties of Diplopia.**—There are two kinds of diplopia, called *lateral* (horizontal) and *vertical*, according as the images are separated laterally or vertically. If, when the images are separated laterally, the right image pertains to the right eye, and the left image to the left eye, the diplopia is designated *simple* or *homonymous;* if the right image pertains to the left eye, and the left image to the right eye, the diplopia is named *crossed* or *heteronymous.* The explanation of these conditions has been given (see Figs. 263 and 264).

C. **Special Symptoms.**—In the following paragraphs the most important symptoms peculiar to paralysis of individual muscles are recorded. For convenience it is supposed that the *right* eye is affected.

1. **External Rectus.**—The following phenomena may be present:

(*a*) *Lateral homonymous diplopia,* the images being side by side and parallel if the eyes are directed on a horizontal level, the distance between them increasing as the test-object is moved to the right—that is, the maximum diplopia is to the right (Fig. 266).

If the test-object is moved to the right and above, and the eyes are directed toward it, the false image (image of the right or affected eye) diverges from the real image (image of the left or unaffected eye). This occurs because, in these circumstances, the movement of the right eyeball toward the temple is limited by the feeble external rectus, and the eyeball fails to come into the position where the inferior oblique has its favorable condition for rotating the vertical meridian outward; hence the vertical meridian remains near to a perpendicular, while that of the sound eye is tilted toward it. There is divergence of the vertical



FIG. 266.—*A*, Position of images in paralysis of left external rectus, and *B*, in paralysis of right externus. The false image is drawn in outline (after Fuchs).

meridians (the false image converges toward the real one) when the eyes are directed downward and toward the right, because the eyeball fails to come into a favorable position to have its vertical meridian tilted toward the nose by the superior oblique, while that of the other eye is tilted toward the temple by the inferior rectus.

(*b*) *Convergent strabismus*, which increases as the eye attempts to follow an object which is moved toward the right, during which it will be noticed that there is *limitation of movement* in this direction.



FIG. 267.—*A*, Position of images in paralysis of left internal rectus, and *B*, in paralysis of right internus. The false image is drawn in outline (after Fuchs).

(*c*) The *secondary deviation* of the sound eye is inward; the *false projection of the field of vision* is to the right side, and the *face is turned* to the *right*—*i. e.*, to the side of the affected muscle.

2. **Internal Rectus.**—There are present:

(*a*) *Lateral crossed diplopia*, the images being side by side and parallel if the eyes are directed along a horizontal level, the distance between them increasing as the test-object is moved to the left, or if the eyes are directed upward—that is, the maximum diplopia is to the left (Fig. 267).

If the test-object is moved to the left and above, and the eyes are directed toward it, the image of the affected eye is lower than that of

the unaffected eye, and its upper extremity inclines toward it; if the test-object is moved to the left and downward, the false image is higher and its lower extremity inclines away from that of the real image. These inclinations occur because, in these circumstances, the left eyeball is placed in a favorable position for one of the oblique muscles to rotate it, while the right eye is not brought in sufficiently for the superior or inferior rectus to exercise its torsion effect; consequently, the vertical meridians diverge on looking upward and converge on looking downward toward the left side.

(*b*) *Divergent strabismus*, which increases when the eye attempts to follow an object moved to the left, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is outward, the *false projection of the visual field* is to the left side, and the *face is turned* to the *left—i. e.*, to the side of the affected muscle.

3. **Superior Rectus.**—There are present:

(*a*) *Vertical crossed diplopia* in the upper field, the images being one above the other, the image of the affected eye being higher than its



FIG. 268.—*A*, Position of images in paralysis of left superior rectus, and *B*, in paralysis of right superior rectus (Fuchs).

fellow and inclined to the left (healthy side), and the vertical distance between them (difference in height) increasing as the test-object is moved upward and to the right—that is, there is maximum diplopia in looking up and to the right (Fig. 268).

If the test-object is moved upward, and to the left, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image is still more inclined toward the sound side, away from that of the other. This occurs because, in these circumstances, the inferior oblique rotates the vertical meridian of the sound eye to the left, while the affected eye, owing to the loss of power in the superior rectus, is unable to deviate its vertical meridian from the perpendicular; therefore the two meridians diverge, but, the diplopia being crossed, the images also diverge.

(*b*) *Downward strabismus*, which increases when the eye attempts to follow an object moved upward, especially upward and outward, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is upward, the *false*

projection of the visual field is too high, and the *face is directed upward and to the right*, or the *head is tilted toward one shoulder*, generally the left.

4. **Inferior Oblique.**—There are present:

(*a*) *Vertical homonymous diplopia* (*sometimes crossed*) in the upper field, the images being one above the other, the image of the affected eye being higher than its fellow and inclined to the right—*i. e.*, to the affected side—the vertical distance between them (difference in height) increasing as the test-object is moved upward and to the left—that is, there is maximum diplopia on looking up and to the left.

If the test-object is moved upward and to the right and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image is still more inclined away from the sound side. This occurs because, in these circumstances, the vertical meridian of the right eye is not tilted toward the temple, owing to loss of power in the inferior oblique, while that of the left eye is tilted toward the nose by the superior rectus, now in its best position for tilting the vertical meridian inward; therefore the two meridians incline toward each other by their upper extremities.



FIG. 269.—*A*, Position of images in paralysis of left inferior oblique, and *B*, in paralysis of right inferior oblique (after Fuchs).

(*b*) The *deviation of the affected eye* is downward and somewhat inward, and is more noticeable when the eye attempts to follow an object moved upward and inward, during which effort it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is upward and inward, the *false projection of the visual field* is too far upward, and the *face is directed upward* and *toward the left*, or the *head is tilted* toward one shoulder.

5. **Inferior Rectus.**—There are present:

(*a*) *Vertical crossed diplopia* in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow and inclined to the right—*i. e.*, to the affected side—and the vertical distance between them (difference in height) increasing as the test-object is moved downward and to the right—that is, there is maximum diplopia on looking down and to the right.

If the test-object is moved downward and to the left, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image inclines still more toward the affected side. This occurs

because, in these circumstances, the superior oblique of the left eye is in its best position for rotating the vertical meridian toward the nose; but the right eye, by reason of its paralyzed inferior rectus, is unable to tilt its vertical meridian to correspond; therefore the vertical meridian of the right eye remains perpendicular, while that of the left eye inclines toward it.

(*b*) *Upward strabismus*, which increases when the eye attempts to follow an object moved downward, especially downward and outward, during which it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is downward and outward, the *false projection of the visual field* is too far downward, and the *face is directed downward* and to the right, or the *head is tilted* toward one shoulder, generally the right.



FIG. 270.—*A*, Position of images in paralysis of left inferior rectus, and *B*, in paralysis of right inferior rectus (after Fuchs).

6. **Superior Oblique.**—There are present:

(*a*) *Vertical homonymous diplopia* (*sometimes crossed*) in the lower field, the images being one above the other, the image of the affected eye being lower than its fellow, and inclined to the left—*i. e.*, to the sound side—the vertical distance between them (difference in height) increasing as the test-object is moved downward and to the left—that is, there is maximum diplopia downward and to the left.

If the test-object is moved downward and to the right, and the eyes are directed toward it, the obliquity of the images increases—*i. e.*, the false image inclines still more toward the sound side. This occurs because, in these circumstances, the vertical meridian of the left eye is inclined toward the left by the inferior rectus, while that of the right eye is not rotated, owing to the feeble superior oblique; consequently, the meridians diverge.[1]

[1] In paralysis of the inferior rectus the diplopia is usually crossed; this feature helps to distinguish it from paralysis of the superior oblique. In both, the image of the affected eye sometimes seems to stand nearer to the patient than the other image. It should be remembered, however, as Mauthner showed, that in paralysis of any one of the obliques a pre-existing exophoria may complicate the case to such an extent as to change "homonymous" into "crossed" diplopia, while in paralysis of the superior and inferior recti pre-existing esophoria may convert "crossed" into "homonymous" diplopia.

(*b*) The *deviation of the affected eye is upward and somewhat inward*, and is more noticeable when the eye attempts to follow an object moved downward and inward, during which effort it will be noticed that there is *limitation of movement* in this direction.

(*c*) The *secondary deviation* of the sound eye is downward and inward, the *false projection of the visual field* is too far downward, and the *face is inclined downward* and to the *left*, or the *head is tilted* toward one shoulder, generally the left.

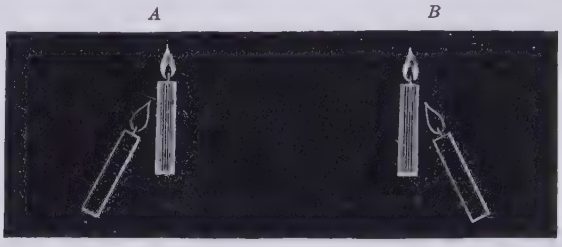


FIG. 271.—*A*, Position of images in paralysis of left superior oblique, and *B*, in paralysis of right superior oblique (after Fuchs).

7. Oculomotor Paralysis.—There are present:

(*a*) *Complete crossed diplopia*, the image of the affected eye being higher than its fellow, and its upper extremity inclined to the right—*i. e.*, to the affected side—the distance between them—*i. e.*, the lateral distance—increasing as the test-object is moved to the left. If the test-object is moved upward the difference in height and the inclination of the false image increase.



FIG. 272.—Double oculomotor palsy (from a patient in Philadelphia General Hospital).

(*b*) *Divergent strabismus* and *limitation of movement* in all directions, except outward and slightly downward.

(*c*) The *secondary deviation* of the sound eye is outward, the *false projection of the field of vision* is to the inner side, and the *face is inclined* toward the *right*, the chin being tipped *upward*. In addition, there are

ptosis, medium dilatation of the pupil which fails to contract to light, and paralysis of accommodation.

Method of Examination and Diagnosis of the Affected Eye.—If the paralysis is complete, there is little difficulty in making a diagnosis by attention to the prominent symptoms which have been detailed. If the condition is one of partial paralysis (paresis), the diagnosis must be based upon an investigation of the double images.

The patient is seated with the head and eyes in the primary position, 4 meters from the test-object (a candle flame or small electric light), and a trial-frame one side of which carries a red glass is placed in position. Hence if diplopia is developed, one image will be yellow and the other red. The lighted candle is then moved from the median line to the right, to the left, upward and down, while the patient follows these movements with his eyes, the head being stationary. By these maneuvers the following facts will be ascertained:

(1) Double images are chiefly seen when the eyes are turned in a direction requiring an action of the affected muscle. (2) The image of the affected eye (false image) is projected in a direction toward which the paralyzed muscle normally rotates the eye. (3) That image is false (image of the affected eye) which travels farther away from the true image (image of the sound eye) when the test-object is moved in the direction of the paralyzed muscle—*i. e.*, the relative distance of the double images increases in these circumstances.

The effect upon the obliquity of the images and their relation to each other of moving the test-object in oblique directions above and below the horizontal plane must next be studied; also whether the images are present in all portions of the field of fixation, or confined to a certain area of it (see also page 606).

Many tables have been prepared to aid in the diagnosis of the affected muscle, and if paralysis of the oblique muscles always produced homonymous or simple diplopia, and paralysis of the superior and inferior rectus muscles always caused heteronymous or crossed diplopia, their construction would be comparatively simple. This, however, is not the case, and it is well known, as has already been pointed out, that the diplopia from paresis of the obliques may be crossed, and that from paresis of the superior and inferior recti homonymous. Hence Duane insists that paralysis of the obliques and of the superior and inferior recti should be diagnosticated from the behavior of the vertical diplopia.

This author divides the twelve muscles moving the two eyes into *three groups* of four each: four moving the eyes laterally, four moving them up (elevators), and four moving them down. Each group is divided into *two pairs*. Thus, the four *laterally acting muscles* are divided into (*a*) a pair of *right turners* (right externus and left internus), and (*b*) a pair of *left turners* (right internus and left externus). The four *elevators* are divided into (*a*) a pair of *right-hand elevators* (right superior rectus and left inferior oblique) and (*b*) a pair of *left-hand elevators* (right inferior oblique and a left superior rectus). The four *depressors* are divided into (*a*) a pair of *right-hand depressors* (right

inferior rectus and left superior oblique, and (*b*) a pair of *left-hand depressors* (right superior oblique and left inferior rectus).

In order to assist in the diagnosis of the affected muscle the following table has been constructed by Dr. Duane which the author has found to be most satisfactory, and which Dr. Duane permits him to insert:

TABLE OF DIPLOPIA IN OCULAR MUSCLE PARALYSIS, ACCORDING TO DUANE

A. There is a lateral (*i. e.*, a homonymous or crossed) diplopia which increases markedly as eyes are carried laterally (to right or left). A laterally acting muscle is paralyzed.
   (*a*) Diplopia increases in looking to the right (= paralysis of a right turner).
      Diplopia homonymous: Paralysis of right externus.
      Diplopia crossed: Paralysis of left internus.
   (*b*) Diplopia increases in looking to the left (= paralysis of a left turner).
      Diplopia crossed: Paralysis of right internus.
      Diplopia homonymous: Paralysis of left externus.

B. There is vertical diplopia which increases in looking up. An elevator is paralyzed.
   (*a*) Vertical diplopia increases in looking up and to the right (= paralysis of a right-handed elevator).
      Diplopia left (*i. e.*, image of right eye above): Paralysis of right superior rectus.
      Diplopia right (*i. e.*, image of left eye above): Paralysis of left inferior oblique.
   (*b*) Vertical diplopia increases in looking up and to the left (= paralysis of a left-hand elevator).
      Diplopia left (*i. e.*, image of right eye above): Paralysis of right inferior oblique.
      Diplopia right (*i. e.*, image of left eye above): Paralysis of left superior rectus.

C. There is a vertical diplopia which increases in looking down. A depressor is paralyzed.
   (*a*) Vertical diplopia increases in looking down and to the right (= paralysis of a right-hand depressor).
      Diplopia right (*i. e.*, image of right eye below): Paralysis of right inferior rectus.
      Diplopia left (*i. e.*, image of left eye below): Paralysis of left superior oblique.
   (*b*) Vertical diplopia increases in looking down and to the left (= paralysis of a left-hand depressor).
      Diplopia right (*i. e.*, image of right eye below): Paralysis of right superior oblique.
      Diplopia left (*i. e.*, image of left eye below): Paralysis of left inferior rectus.

To illustrate the practical working of the table the following example is quoted: The patient with a red glass before the right eye is directed to observe a candle which is moved in all directions in his field of fixation. If the patient has single vision when he looks down, but has vertical diplopia when he looks up, paralysis of an elevator is inferred. The vertical diplopia increases greatly when he looks up and to the right, and diminishes to almost nothing when he looks up and to the left. The paralysis must affect a right-hand elevator (right superior rectus or left inferior oblique). The red image is higher (left diplopia = right eye below). The paralysis must affect the right superior rectus. If it had been the left inferior oblique, the red image would have been the lower; and if it had been either the right inferior oblique or the left superior rectus, the vertical diplopia would have increased not when the patient looked up and to the right, but when he looked up and to the left.

Classification and Site of Lesions.—Paralysis of the ocular muscles may be due to a lesion which resides within the cranium (intracranial), or without it (extracranial), and may be situated at any point on the oculomotor apparatus from the cortex of the brain to its termination in the muscle. The extracranial lesions reside within the orbit (intra-orbital or orbital), and affect the muscles themselves, or their nerve supply. The intracranial lesions may reside within the cerebrum (intracerebral), and affect the cortex, subcortex, nerve-nuclei, nerve-roots, and connecting fibers, or outside of the cerebrum (extracerebral), and affect the nerve trunks at the base of the brain (*basal paralysis*).

Etiology.—The influence of the lesion may be a *direct* one in that the nerves themselves or their origins are implicated by an inflammatory or degenerative process, or an *indirect* one through the agency of surrounding conditions, for example, meningitis, tumors, vascular alterations and cerebrospinal disease, ataxia, paretic dementia, etc. Moreover, one and the same cause may be effective in all of these different ways, for example, syphilis.

Among the conditions residing in the orbit which produce paralysis of the exterior ocular muscles, *intra-orbital* or *orbital palsies*, are cellulitis, tenonitis, periostitis, tumors, metastatic carcinomatous nodules (Elschnig), hemorrhage, and affections of the paranasal sinuses by reason of a transfer of infection to the tissues of the orbit (page 679). Abducens palsy has followed nasal trauma and infection (Ewing, Sluder), and the author has seen complete isolated paralysis of the internal rectus immediately after the removal of a polypus from the nose. Other causes are *injuries*—fracture of the orbital walls, penetrating wounds whereby the muscle is divided or torn, foreign bodies, etc. Isolated palsy of the inferior oblique is most frequently traumatic (S. B. Marlow), and orbital muscles may be injured during an operation on the orbit (removal of a tumor) or on the sinuses (Killian's operation), especially the superior oblique. Certain types of chronic external bilateral ophthalmoplegia (ptosis alone, or ptosis with implication of other orbital muscles; also in some of them orbicularis palsy), according to Treacher Collins, may be due to a primary degeneration of muscle fibers (*abiotrophy*). Duane, therefore, classifies them in the orbital muscle group of palsies.

Syphilis, taking into consideration its manifold ways of attack, is responsible for about one-half of the cases of exterior ocular muscle palsies.[1] They may be due to an inflammation or gummatous change affecting the nerves at the base of the brain or in the orbit, or to disease of the nuclei of the nerves or of the brain in their immediate vicinity, or to lesions in the third ventricle, the aqueduct of Sylvius, or the fourth ventricle. (See also page 619.) Syphilitic paralysis is generally one of the later manifestations, but it has been noted as early as, or even

[1] The frequency of syphilis as a cause of ocular muscle palsy has been variously estimated from 18 to 60 per cent. Modern methods of determining the etiologic factor in ocular muscle palsies, that is, the Wassermann test, etc., will improve the accuracy of later statistical information.

earlier than, the sixth month after the primary infection, particularly in the form of *ptosis*. Syphilis attacks the oculomotor most frequently, next in order the abducens (13 per cent. [Igersheimer]), and least frequently the trochlearis, isolated palsy of which is very rare. Paralysis of the ocular muscles from *inherited syphilis* is comparatively unusual, something over 30 cases being recorded in the literature (Igersheimer).

Other causes of palsy of individual exterior eye muscles, some of which at times occasion *central*—that is, *nuclear*—lesions, and at other times act *peripherally*, are rheumatism, gout, diabetes, whooping-cough, influenza, dengue fever (Barkan), typhoid fever, herpes zoster, and certain toxic agents—for example, lead, alcohol, tobacco, gelsemium, conium, chloral, and ptomains.

Not uncommonly the etiologic factor in palsies apparently peripheral in origin is vague, or not discovered. The so-called rheumatic palsies are of this nature; they are often attributed by patients to a chill, or to "catching cold." The external rectus is commonly involved, and generally the prognosis is good; but evidently, as Mauthner pointed out, in some instances these palsies may be followed years later by tabes, disseminated sclerosis, and paretic dementia. Although diphtheria usually affects the ciliary muscle, it may attack one or more of the exterior muscles, generally the external rectus. The condition may be bilateral. Rarely complete ophthalmoplegia occurs. Otitis media, with isolated paralysis of the abducens on the corresponding side and intense unilateral headache, is known as *Gradenigo's symptom-complex*, and usually requires operation from the otologic standpoint for its relief.

The diseases and lesions which attack the nerves at the base of the brain, and thus occasion the so-called *basal palsies*, are hemorrhage, meningitis, both simple and tubercular, particularly the latter, abscess, for example, in connection with middle-ear disease, sinus disease, aneurysm, thrombosis of the cavernous sinus, pituitary body disease, syphilis, and tumors, for example, in the middle fossa of the skull. *Spinal anesthesia* may be followed by palsy of the exterior ocular muscles (5 times in 2000 anesthesias [Reber]), the external rectus being most frequently involved. This paralysis probably depends upon a toxic reaction in the meninges.

A number of paralyses of the exterior ocular muscles are associated with locomotor ataxia, paretic dementia, disseminated sclerosis, and bulbar paralysis. Tabetic paralysis is often transitory in its nature; it may be accompanied by the pupillary changes characteristic of this affection. Relapses are frequent; the palsy may be permanent. Sherrington has demonstrated that the third, fourth, and sixth nerves contain afferent fibers which are not derived from the fifth nerve, and concludes that tabetic ocular palsies are probably due to implication of these afferent fibers and not to the implication of the efferent fibers of the ocular nerves. Paralyses of the orbital muscles of *cerebral* origin may result from degenerative, hemorrhagic, or neoplastic lesions affecting the cortex of the brain, the corticopeduncular region, the nuclei of the nerves, or the nuclear fibers. Lesions of the cortex do not cause isolated par-

alyses (except occasionally *ptosis*); they are concerned with conjugate palsies (page 623). *Lethargic encephalitis*, a disease very prevalent in recent years, has conspicuously among its manifestations paresis and paralysis both of the exterior and interior ocular muscles. In mild type the muscle palsies subside with the disappearance of the other symptoms. In severe cases there may be complete *ophthalmoplegia;* in other cases individual muscles may be affected. The palsy of the ciliary muscle may be long continued. Nystagmus is not infrequent. (For further consideration of this subject see page 645.)

Injury as a cause of ocular muscle palsy in the orbit has been mentioned. Fracture of the base of the skull often produces paralysis of eye muscles, their supplying nerves having been damaged as they pass along the base of the brain. The palsy may develop secondarily from basal meningitis, abscess, or nuclear degeneration.

Anomalies of the exterior muscles may occur, depending upon their abnormal insertion. Entire absence of a muscle has been noted. Occasionally cases of orbital muscle palsy have been attributed (without any convincing reason) to various so-called *reflex disturbances.*

Ophthalmoplegia.—Although the term "ophthalmoplegia" might with perfect propriety be used to describe all the ocular muscle palsies, it is generally reserved for that class of paralyses of the orbital muscles due to disease of the nuclei of the third, fourth, and sixth nerves. Paton quotes Mauthner's definition of an ophthalmoplegia as a condition in which either the muscles of both eyes are affected, especially if they are supplied by different nerves, or if only one eye is involved, more than one nerve of supply must be affected. He also emphasizes the fact that there is no sharply defined limit between lesions affecting nuclei, that is, group of cells, nerve roots or association fibers.

Ophthalmoplegia may be divided into *acute ophthalmoplegia* or *acute nuclear palsy*, and into *chronic ophthalmoplegia* or *chronic nuclear palsy*. When it so happens that the intra-ocular muscles alone are affected, the term *interior ophthalmoplegia* is sometimes employed (page 625), and when the exterior muscles alone are affected, the term *exterior ophthalmoplegia.* When both sets of muscles are involved, the term *total ophthalmoplegia* is appropriate.

Acute ophthalmoplegia is characterized by a rapid paralysis of all ocular muscles, often associated with fever and convulsions. Many of the cases have proved to be fatal. They occur with hemorrhage in the region of the nuclei, or as an acute hemorrhagic polio-encephalitis, the primary cause being tuberculosis, syphilis, ptomain-toxemia (botulism), or poisoning from alcohol or sulphuric acid. Acute ophthalmoplegia may be associated with acute poliomyelitis, with bulbar palsy, or with facial palsy. Certain poisons—for example, nicotin, lead, and carbon monoxid—may cause an ophthalmoplegia which is not fatal, or, at least, not necessarily fatal, and the same is true of one type which is associated with certain constitutional and infectious diseases—for instance, diabetes, diphtheria, syphilis, lethargic encephalitis, and

influenza. Among the infections and poisons which have been named (also mentioned on page 619), *lethargic encephalitis* and *botulism*, especially the former, have attracted a large measure of attention. In lethargic encephalitis, independently of somnolence, "mask face," facial and other palsies, etc., disturbances of the oculomotor functions are conspicuous, and may appear in mild or severe form. The external rectus may be the only muscle involved (sometimes the superior oblique), or there may be complete paralysis of the third nerve, and even double complete ophthalmoplegia. The ciliary muscle is early affected, and almost always the last one to recover.

In botulism (including fish, sausage, sour cheese, and meat poisoning) ptosis and paralysis of the ciliary muscle are common; other branches of the third nerve may be involved; also the sixth and fourth. Usually the prognosis is good, but fatal outbreaks of *botulism* caused by eating contaminated olives have occurred; the toxin depended upon type A of the *Bacillus botulinus*.

Transient bilateral ophthalmoplegia has been described, the symptoms developing rapidly and disappearing completely after one or two months.

Chronic ophthalmoplegia is characterized by loss of power in one or more eye muscles, which may gradually increase until every muscle is paralyzed. Sometimes the levator escapes; indeed, ptosis may be absent. The disease may be *stationary* or *progressive*. It is not always symmetric; it may be unilateral. Chronic ophthalmoplegia may follow an acute palsy, the lesions of which have started chronic degenerative changes; it may appear as a *congenital* and occasionally *hereditary* affection, usually in the form of bilateral ptosis. Thus, A. A. Bradburne observed ptosis, with almost complete loss of ocular movement, in a family where this affection has been present in five generations. Sometimes epicanthus complicates the ptosis. Wilbrand and Saenger have described a type of ophthalmoplegia characterized by progressive bilateral paralysis of the external eye muscles unassociated with other signs of disorder of the nervous system, with ptosis usually as the first symptom. It begins in infancy or childhood (occasionally at a later period). It is extremely slow in its progress and many years (thirty or more) may elapse before the termination in complete double ophthalmoplegia takes place. It is not certain whether this disease should be regarded as a separate clinical entity. Cases of this character, or similar to them, have been recorded by a number of observers (Wilbrand and Saenger collected 32, and Bradburne, Beaumont, and recently McMullen and Hine have reported others). It is possible that they may be due to lack of development of the nuclei, as Hine and McMullen point out, but also to primary atrophy of the muscles, as previously mentioned (page 617). Ophthalmoplegia may be associated with locomotor ataxia, paretic dementia, progressive muscular atrophy, chronic bulbar paralysis, and disseminated sclerosis. The underlying constitutional condition of chronic ophthalmoplegia may be syphilis and sometimes tuberculosis. The disease is essentially chronic, and may last

for years. It is more common in males than in females, and is more serious in children than in adults.

The intra-ocular muscles usually escape, but this is not always the case. If they escape there is presumptive evidence that the origin of the trouble is nuclear, but, as Mauthner pointed out, it is not a characteristic sign, and a partial palsy may be peripheral. Siemerling concludes that nuclear disease may be inferred from exterior ophthalmoplegia, if it is not maintained that nuclear palsy must manifest itself as an exterior ophthalmoplegia. In general terms the lesions are degenerative, inflammatory, or hemorrhagic. According to Siemerling, the pathologic states underlying progressive paralysis of the ocular muscles may reside in nuclear disease, in degeneration of the muscles and of the nerve-trunks, the nuclei being intact, and in interruption of the conducting power of the intramedullary roots, muscles, nerve-trunks, the nuclei being uninvolved.

Recurrent Oculomotor Paralysis (*Ophthalmoplegic Migraine*—Charcot).—The symptoms of this comparatively rare affection are violent unilateral headache, nausea, vomiting, slight fever, and usually paralysis of the third nerve on the same side as the pain. The attacks come in periodic crises, and the disease may last from several days to long periods of time. Occasionally the paralysis becomes permanent. The lesion is probably one involving the root of the third nerve. *Recurrent paralysis of the abducens* has been observed.

Congenital Paralysis or Deviation.—Congenital palsies of the exterior ocular muscles, usually stated to be uncommon, are comparatively frequent if slight deviations are taken into account. They have been particularly well studied by Duane.[1] According to him, insufficiency of the superior rectus, with fixation with the paretic eye and secondary deviation (upshoot) of the other eye, is not uncommon. A striking feature is that when the fixing eye moves outward, the other shoots spasmodically up and in (spasmodic action of the inferior oblique). Other groups described by Duane are palsy of the superior rectus with fixation with the non-paretic eye; slight and marked insufficiency of the inferior rectus; insufficiency of the inferior and superior oblique and combined insufficiencies of the vertical and lateral muscles. For operative treatment see page 782. Congenital palsy of one or both externi is not uncommon, and may be associated with retraction movements (see next paragraph). The symptoms of congenital palsy usually are: head-tilting, diplopia, vertigo, and asthenopia. The patients nearly always close the defective eye.

Retraction Movement of the Eyeball, Associated with Congenital Deficiency of Abduction.—Certain cases of congenital deficiency of movement of the eyeball are characterized by all or some of the following symptoms, which have been thus summarized by Duane: Usually complete, occasionally partial, absence of outward movement of the affected eye; partial defect of inward movement of the affected

[1] Transactions of the American Ophthalmological Society, vol. xii, Part iii, 1911.

eye; retraction of the affected eye into the orbit when it is adducted; a sharp, oblique movement of the affected eye up and in or down and in when it is adducted; partial closure of the eyelids of the affected eye when it is adducted; and paresis or, at least, marked deficiency of convergence, the affected eye remaining fixed in the primary position, while the sound eye is converging. Enophthalmos may be present in primary position of the eyelids (A. Lutz).

The affection is undoubtedly congenital. Females are more usually affected than males. The affection has been explained by assuming that the externus is replaced by an elastic or inelastic strand of connective tissue, or that a faulty insertion of the internus causes it to act as a retractor. The oblique upward and downward movements of the eye observed in many of these cases during adduction are attributed by Duane to a spasmodic action of one of the obliques. Operative procedures have been suggested for cosmetic purposes—namely, tenotomy of the retractor muscle and fixation of the globe in the opposite position (Wolff); or transplantation of the split tendon of the external rectus to superior and inferior rectus respectively.

It is often difficult to ascertain whether the paralysis is *central* or *peripheral* in its origin. The differential diagnosis must be made by examining into the completeness of the paralysis and the existence of complications or associated symptoms. Peripheral palsies are more apt to be isolated and complete; those of central origin are often associated with other symptoms indicative of intracranial mischief.

Relative Frequency of Paralysis of the Orbital Muscles.—Paralysis of the abducens (external rectus) is met with most frequently, the next in order of frequency being unilateral paralysis of the oculomotor. After these come paralysis of the superior oblique, inferior rectus, superior rectus, internal rectus, and inferior oblique. However, statisticians differ exceedingly on these points—*e. g.*, Duane ranks the superior rectus next to the external rectus.

Prognosis.—This depends upon the cause of the palsy. Some cases of peripheral paralysis, especially those depending upon syphilis and rheumatism, are readily amenable to treatment; in others not only is the paralysis incurable, but the lesion which creates it may be a fatal one. Hence the importance of trying to ascertain the character and situation of the lesion which produces the palsy.

Treatment.—In syphilis the usual remedies are indicated, and in many instances good results follow very large doses of iodid of potassium. Massive doses are often tolerated, and even if the paralysis has existed for a long time, cure may ultimately result. Salvarsan or arsphenamin, in the author's experience, is of comparatively little service in luetic palsy of the exterior ocular muscles, that is, in cases of long standing. In those cases of ocular muscle palsy which develop, often the external rectus, in patients who are the subject of various forms of chronic rheumatism and myalgia, in addition to iodid of potassium, salicylic acid is useful, especially in the earlier stages. Disturbances of metabolism, as, for example, gout and diabetes, require the usual treatment.

The various causes which have been mentioned furnish the indications for other treatment. Care should always be taken to examine the nasal accessory sinuses for sources of infection and the intestines for the evidences of toxic products.

The great annoyance which is produced by the double images may be remedied by covering the affected eye with a piece of ground glass, which is mounted in a spectacle-frame. If the patient is ametropic, his correcting lens for the opposite eye may be placed in the same frame.

Sometimes prisms may be worn which fuse the double images. The rules for adjusting prisms are given on pages 643 and 644.

Electricity may be tried, the great difficulty being in passing the current through the muscle. Ordinarily, one pole—the cathode—is placed upon the closed lid, while the other is put upon the temple. Usually, a current of more than 3 milliamperes is unbearable. This is especially true if the pole is placed directly upon the sclera, the eye first having been cocainized. Very disagreeable flashes of light will usually take place if a current of more than 1 or $1\frac{1}{2}$ milliamperes is employed. If faradism is tried, a very weak current should be selected.

Finally, after all other means have failed, tenotomy of the contracted muscle has been resorted to, but usually is successful only if it is combined with advancement of the paralyzed muscle. The best results are obtained in the lateral muscles. In case of an injured muscle—that is, one torn from its insertion—it would be proper to find the ends of the divided muscle and stitch them together. Indeed, advancement of a severed muscle is quite possible long after the accident or after a too extensive tenotomy. Operation for paralysis of the superior oblique to neutralize the diplopia would require tenotomy of the inferior rectus of the opposite eye, or advancement of the inferior rectus of the paralyzed eye (Landolt). The former procedure is preferable. So, too, paralysis of an inferior oblique would require, to relieve diplopia, tenotomy of the superior rectus of the opposite eye (see also page 637).

Associated Ocular Paralyses (*Conjugate Deviation*).—Sometimes the eyes cannot make certain movements in which they are usually associated, although the directing power of the muscles may be unimpaired when they exercise their function in a different association. In other words, there is paralysis of movement and not of the muscles supplied by a given nerve (*paralysis of ocular gyration*). Thus the internal recti may be unable to draw the eyes together in the act of convergence, although they may act normally in helping to move the eyes from side to side; or there may be loss of the synchronous lateral movement of the external rectus of one eye and the internal rectus of the other (*conjugate lateral paralysis*), although convergence is normal; or the upward or downward movements of the eye may be lost. Lesions affecting the centers for combined movements may produce such phenomena, the majority of these palsies being due to a lesion involving the abducens nuclei. According to Spiller, paralysis of lateral associated movement may be caused by a lesion of the posterior longitudinal bundle or of the sixth nucleus; symmetric disease of the nuclei of

the affected nerve explains some cases in which the upward and the downward movement are lost. The lesion may be in or near the corpora quadrigemina. Paralysis of associated downward movements is an unusual condition. J. W. White attributes it to a lesion of the oculomotor nuclei or to one located in the corpora quadrigemina. Holmes and Sargent have described disturbance of ocular movement with injury of the superior longitudinal sinus; in one group temporary palsy of associated conjugate movements occurred, attributed to implication of the posterior part of each frontal convolution. Typical spasmodic conjugate deviation may be caused by hysteria, and this neurosis may also originate palsy of associated parallel movements.

In apoplexy, if the head is drawn from the paralyzed side and the eyes are also turned to the sound side, the condition is called *conjugate deviation of the head and eyes*. The rule is, according to Prevost, that in lesions of the hemisphere the eyes are turned toward the lesion and away from the paralyzed side, but in lesions of the mesencephalon they are turned away from the lesion and toward the paralyzed side. Should there be unilateral convulsions, with the eye turned toward the convulsed side, there is an irritative lesion in the hemisphere, but if the head and eyes are turned away from the convulsed side, there is an irritative lesion in the mesencephalon (Landouzy).

Divergence Paralysis.—This condition which is usually sudden in onset manifests itself by homonymous diplopia and convergent strabismus when the eyes are fixed upon a distant point. As the test-object approaches the patient, and especially on lateral fixation, there is diminution of the convergent strabismus and the diplopia, and finally a point may be reached where there are single vision and orthophoria, while within this limit there may be exophoria. Cases of this character have been described as secondary to an abducens paralysis, and also ascribed to spasm of convergence and to paralysis of a supposed divergence center. Berry believes the correct diagnosis of this condition to be *spasm of convergence* and not paresis of divergence. Duane, however, maintains that the only satisfactory explanation of the phenomenon is that it is due to a *paralysis of divergence*. It occurs at all ages, and more often in conjunction with hyperopia than myopia. He suggests that a lesion near the two abducens nuclei would cause this condition. As causes Alger records cerebral hemorrhage, nephritis with high tension, tabes dorsalis, and multiple sclerosis; it has been observed as an unusual complication of epidemic encephalitis (Dunnington, Zentmayer, Holden).

Convergence Paralysis.—This, as an extreme variety of convergence insufficiency, is referred to on page 639. The symptoms are: crossed diplopia, divergent strabismus, which increases as the test-object is made to approach the eye, no increase of diplopia either to the right or left, and normal rotation of each eye outward and inward. It has been observed in various central nervous disorders, and notably in locomotor ataxia and disseminated sclerosis; also in lethargic encephalitis.

Paralysis of the Interior Ocular Muscles.—Under the general term *cycloplegia* are included the cases of paralysis of the ciliary muscle. These may or may not be associated with dilatation of the pupil.

If the ciliary muscle is paralyzed, the chief symptom is loss of accommodation, precisely as it occurs after the instillation of a mydriatic. The loss of accommodation may be *complete* or it may be *partial;* that is, one or more diopters of the entire amount which is normal at the patient's time of life may still remain. After the fiftieth year it is difficult to detect cycloplegia.

It occurs from a lesion in the trunk of the oculomotor nerve or in the anterior part of its nucleus (consult also Oculomotor Palsy and Ophthalmoplegia). Unilateral cycloplegia is said to be possible under the influence of disease of the ciliary ganglion. Paralysis of accommodation may be caused by affections of the nervous system, infectious diseases, and by intoxications. A very common cause of double paralysis of the ciliary muscle is diphtheria. Cycloplegia is also occasioned by spinal disease, by diabetes, by disease of the accessory sinuses, by various focal infections, by so-called auto-intoxication, by disorders of metabolism, by traumatism (usually then associated with mydriasis), by prolonged eye-work, by mumps, by tonsillitis, and frequently by acquired syphilis, and is often associated with paralysis of the sphincter of the iris. Inherited syphilis is a rare cause of paralysis of accommodation. Paresis of the ciliary muscle is common after certain fevers—for example, typhoid fever. Various ptomains, toxins, fish, and meat poisonings may cause both paresis and paralysis of the ciliary muscle. Congenital interior ophthalmoplegia is a rare anomaly (Duane). Toxic and traumatic cycloplegias are usually peripheral; syphilitic and parasyphilitic varieties may be peripheral, basal, and nuclear in origin.

Under the general term *iridoplegia* are included the conditions which occur when there is loss either of the direct or of the associated action of the iris, due to paralysis of its sphincter. The chief symptom is connected with changes in the action of the pupil. The condition may or may not be accompanied by paralysis of the ciliary muscle. The various pupillary changes have been discussed in Chapter II, page 56. Consult also page 621.

Concomitant Strabismus or Squint: Heterotropia.—This form of strabismus is characterized by the power of the squinting eye to follow the movements of the other eye in all directions, the angle of squint always maintaining the same size.

Varieties of Concomitant (*Comitant*) **Strabismus.**—The chief deviations of squinting eyes, as already given, are *convergent strabismus*, or *esotropia; divergent strabismus*, or *exotropia;* and *vertical strabismus*, or *hypertropia*. Concomitant squint may be *periodic* or *constant*. The latter variety is divided into *monocular squint*, that is, in ordinary circumstances the same eye always deviates when the other eye is used for fixation, and *alternating squint*, that is, either eye is used indifferently for fixation. Lateral squint is usually associated with upward deviation. It is probable that at first squint is generally periodic,

but with repeated recurrences, as Priestley Smith expresses it, the suppression of the deviating image becomes confirmed, and the squint becomes continuous. The order of events, according to Duane, is as follows: a child with a decided degree of hyperopia or astigmatism, when he begins to use his eyes for close observation, develops a *spasmodic esophoria* (*convergence-spasm*); later, binocular vision being impossible, *periodic squint appears* (diplopia may be detected); still later binocular fixation is lost and the squint becomes *continuous;* still later divergence insufficiency is added and, finally, muscular changes arise, that is, rotation inward is excessive, rotation outward reduced. The average age for convergent squint to begin is three and four-tenths years, although it is often noticeable during the first year of life. Squints occurring after five years are apt to be alternating, in which case excellent vision exists in each eye. According to Duane, a slight vertical congenital deviation may be the starting-point of a progressive lateral deviation, which is unnoted until the child is five or six years of age.

Causes of Concomitant (*Comitant*) **Strabismus.**—The etiology of strabismus has occasioned much discussion, and even at this time is not a settled question. In general terms, the factors which have been considered important in the causation of squints may be summarized as follows:

1. Disturbance of the relation between accommodation and convergence by errors of refraction.

2. Inequality in the vision of the two eyes, or amblyopia of one eye, which removes the natural stimulus of diplopia to exact convergence.

3. Disturbances of innervation and defective development of the fusion faculty.

These causes of squint are somewhat elaborated in the succeeding paragraphs.

1. *Disturbances in the Relations of the Functions of Accommodation and Convergence.*—The relation between these two functions has been previously described (see page 47). Some latitude of movement is possessed by each function separately, but a limit to the independent exercise of either function exists beyond which neither function can operate alone. Thus, a hyperopia of 6 D would require an accommodation of 6 D to neutralize it, the visual lines being parallel. This is rarely possible; some meter-angles of convergence will usually accompany the accommodative effort. The point of convergence is then nearer than the point accommodated for, constituting a convergent squint. Hyperopia is, therefore, frequently accompanied by convergent squint.

In contrast to this, a myope of 10 D requires 10 meter-angles of convergence to see at his far point of vision, that is, the point at which he can see with relaxed accommodation. This is not usually possible, because the enormous convergence necessary to see at this point is too severe a strain; consequently, the visual lines intersect at a greater

distance than the point for which they are accommodated, and binocular vision is abandoned. The eyes, left to the preponderating forces, assume the direction seen during sleep and deep anesthesia—viz., divergence. Myopia is, therefore, frequently accompanied by divergent squint.

Sometimes individuals possess or acquire unusual power in developing one or other of these two functions. Thus, the hyperope may develop his accommodation sufficiently to equalize the disparity in the refraction and thus avoid squinting. The myope may also develop his convergence beyond the usual amount so as to prevent divergence. Hence all hyperopes do not have convergent squint; neither do all myopes have divergent squint.

2. *Inequality in the Vision of the Two Eyes, or Amblyopia of One Eye, Which Removes the Natural Stimulus of Diplopia to Exact Convergence.*—Amblyopia of the squinting eye is present in a large proportion of the cases of concomitant convergent strabismus, or, more accurately, the amblyopia of the squinting eye exceeds that of the other. Whether this amblyopia is a cause or a consequence of the squint has given rise to two theories. According to one theory, advocated by Donders and others, the squint causes the amblyopia which depends upon a loss of vision due to habitual suppression or to lack of use of the squinting eye—*amblyopia exanopsia*—or, according to Hirschberg's terminology, *amblyopia exablepsia.* According to the other theory, advanced by Schweigger, the amblyopia is a congenital defect which precedes and causes the squint. Priestley Smith points out that all eyes are amblyopic at birth, and reach the normal standard of vision only after several years. If strabismus is established before this standard is attained, further visual progress of the squinting eye is likely to be hindered or even arrested. In general terms it seems that an amblyopia from disease should be recognized as an etiologic factor.

Ophthalmoscopically these amblyopic or so-called "neglected eyes" may be entirely normal, or there may be at times distinct changes in and around the nerve-head and in the macula. Central scotomas and contraction of the visual field are sometimes demonstrable, as the author has shown, and in such circumstances these eyes are not susceptible of improvement in vision (see also page 571).

An amblyopia which removes the stimulus of diplopia to exact convergence may also include cases in which the visual acuteness is diminished by refractive differences in the two eyes, one eye being greatly inferior to its fellow by reason of a high degree of hyperopia or myopia, with or without astigmatism, by opacities in the media of one eye (especially corneal opacities), by congenital cataract, and by complete blindness. The failure to recognize diplopia causes the visual axes to vary considerably either toward convergence or divergence, without appreciation of this on the part of the patient. If the eyes are hyperopic, they are apt to converge; if myopic, to diverge. Numerous cases of squint exist without amblyopia, and the refraction of both eyes may be equal.

3. *Disturbances of Innervation and Defective Development of the Fusion Faculty.*—According to Hansen Grut, "convergent strabismus originates and continues as the result of an *innervation* which effects in the interni a shortening exceeding in amount that which is desirable. Divergent strabismus is the expression for a relaxation of convergence innervation, which permits the eye to take up its anatomic position of rest." According to Priestley Smith, "convergent strabismus is a disorder of innervation in which the visual centers fail to control the act of convergence, which is degraded and becomes automatic. It is excited by the act of accommodation and is excessive because uncontrolled. The failure of control depends largely upon faulty development of the visual apparatus. Hyperopia, when of considerable degree, predisposes to strabismus by demanding an abnormal effort of control. The disorder is confirmed and perpetuated by suppression of the function of the squinting eye."

According to Claud Worth, "when the fusion faculty is fairly well developed, neither hyperopia, anisometropia, nor heterophoria can cause squint. In fact, then, nothing but an actual muscular paralysis can cause an eye to deviate, in which case the resulting diplopia is intolerable. Sometimes, however, owing to a congenital defect the fusion faculty develops later than it should, or it develops very imperfectly, or it may never develop at all. Then, in this case, there is nothing but the motor co-ordinations to preserve the normal relative directions of the eyes, and anything which disturbs the balance of these co-ordinations will cause a permanent squint. Thus, the essential cause of squint is a defect of the fusion faculty." The provocation in the presence of this fundamental cause to squint may be supplied, he believes, by various conditions—for example, hyperopia, anisometropia, heterophoria, amblyopia of one eye, certain eruptive fevers and infections (whooping-cough, especially if hyperopia in any degree is present), violent mental disturbance, and hereditary influence. The influence of heredity in squint is an important matter, and Mr. Worth believes, and this certainly is in accord with the author's experience, that a history of heredity can be obtained in fully 50 per cent. of the cases. Therefore it is important carefully to investigate early in life the eyes of children whose parents or grandparents have squinted. Percival T. Hay's recent investigation of the personal history of squinting children has led him to the conclusion that measles, whooping-cough, scarlet fever, and bronchopneumonia should be regarded as important etiologic factors.

A predisposition to strabismus may arise on account of the size and shape of the eyeball and orbit. A narrow, horizontal diameter of the face might predispose to convergent strabismus, or an unusually broad diameter to divergent strabismus. These conditions may coexist with hyperopia and myopia. A very short eyeball, flattened in its anteroposterior direction, by its greater facility of movement would render convergence easier; the opposite condition—namely, elongation of the anteroposterior axis of the eyeball—would render this movement

more difficult. An unusual value of the *angle gamma* might create a disposition to squint by disturbing the relation between convergence and accommodation. At one time disparity in the length, thickness, and tension of opposing muscles was regarded as an important factor in the development of squint.

Single Vision in Concomitant (*Comitant*) **Strabismus.**—Diplopia is rarely noticed in comitant convergent strabismus, because the deviating eye involuntarily suppresses the image, or else has learned to disregard it.

Suppression of the image is not, however, habitually permanent, or suppression does not extend over the whole visual field, and many patients can be made conscious of diplopia if a red glass or cobalt glass is placed before one eye and a prism before the other. When the squint is very large it may be necessary to correct the greater part of it with prisms before diplopia is manifest. If prisms and the red glass fail, Schweigger's test is as follows: A flame is placed to one side of and behind the squinting eye, and its image is thrown into this eye with a plane glass held close to it. When the reflex reaches the center of the pupil the patient sees it and can describe its relation to the image of another flame observed by the fixing eye at a distant point. With high degrees of amblyopia it may be impossible to produce diplopia.

In comitant divergent squint, especially of low degree, and in the convergent strabismus of myopes, diplopia is not uncommon; also in moderate degrees of convergent strabismus and in the residual squint after tenotomy. Referring to the nature of diplopia in comitant strabismus Claud Worth thus expresses himself: The subject of squint with diplopia sees with his deviating eye a faint eccentrically placed image of the object to which the fixing eye is directed, and suppresses the image of the object which lies in the axis of the deviating eye—*i. e.*, he sees two images of the same object, but not two different objects.

Sometimes after operation, as was first noticed by von Graefe, the *diplopia* is anomalous or *paradoxic*, as it is called—that is, there is crossed diplopia with convergent squint. Javal observed and studied the same phenomenon in strabismic patients upon whom no operation had been performed. It has been explained on the theory that there has been developed in the squinting eye a spot identical with the macula lutea of the straight eye, or that there has been developed what has been named a *vicarious fovea*. According to Tscherning, in certain cases of strabismus a period may be reached after operation when the patient localizes with reference both to the new and the old fovea, the result being *binocular triplopia*, a name given by Javal to the phenomenon. Verhoeff thinks paradoxic diplopia "is due not to the development of a new system of corresponding points, but to an absence of any such system whatever, so that when diplopia is produced, each eye localizes its image with regard to itself alone and hence more or less correctly."

Measurement of Strabismus.—1. Squint may be measured approximately by the deviation inward of the pupil of one eye while the other

eye fixes an object. The pupil being situated 10.5 mm. in advance of the center of rotation, its deviation inward or outward, measured on a rule, represents the tangent of the angle of the squint. A deviation of 1 mm. represents a squint of 5°. For this purpose an ordinary rule divided into millimeters may be employed, or a specially devised instrument curved to adapt itself to the curve of the eyeball and known as a *strabisometer*.

If diplopia is present, as Landolt has shown, it permits an accurate determination of the angle of strabismus. The procedure may be as follows:

> Upon a wall of the consulting room, in a horizontal line, and so as to be on a level with the eyes of the patient, who is about 3 meters from the wall, are permanently marked out tangents of angles of 5° each, as seen from the place where the squinting eye is. Exactly opposite to the squinting eye is 0°, while toward the right and left the points are marked up to 45° or more. The flame of a candle being held at 0°, and one eye of the patient being covered with a red glass, he is called on to indicate the position of the image belonging to the squinting eye, and the number on the wall which corresponds to this gives the angle of the strabismus.

In these circumstances the degree of prism necessary to fuse the double images may be used to measure the squint.

2. *Angular Method.*—The perimeter may be employed to measure squint with reasonable accuracy, although Worth condemns the method because it takes no account of the angle gamma. Landolt thus describes the method:

> The deviating eye, *R*, is placed at the center of the graduated arc of the perimeter, *P–P*, the arc lying on the plane of the deviation. The patient is then required to fix with *his two eyes* a distant object, *A*, situated at the central radius, *R–o–A*. This is the direction which the deviating eye should have in the normal condition. The point *n*, to which the eye in reality is directed, should now be determined; the angle *o–R–n*, formed by the deviating visual line *n*, with the normal line of fixation *A–o–R*, is the *angle of the strabismus*. In order to obtain this direction (*i. e.*, the point *n* at which the eye is directed) it would be necessary only to determine the visual axis. As this is not an easy matter, it is sufficient in practice to be contented with the optical axis; this differs from the former only by the angle gamma, which, in comparison with the large angle of the strabismus, may be neglected (see page 42). The flame of a candle is moved along the arc of the perimeter until its reflection is in the center of the pupil. This will occur when the flame is at *n*. The optical axis has now been found, and the size of the angle of strabismus may be read off.

Priestley Smith's Tape Method.—This is a very good method, although it is not very readily applied to young children. Worth describes it as follows: "A string 1 meter long has a ring at one end. To the ring is attached a graduated tape. The tape has a weight at its other end. The patient holds the free end of the string against his temple. The surgeon puts the ring on a finger of one of his hands, in which he holds an ophthalmoscope mirror. The tape is allowed to slide between the fingers of the other hand, the weight keeping the tape taut. The patient is first told to fix the mirror, while the light of a lamp is reflected into the fixing eye. The position of the image of the mirror on the cornea of the fixing eye is noted. The light from the mirror is now thrown on to the deviating eye, and the patient is directed to look at the surgeon's tape hand. This is moved horizontally till the position of the image of the mirror on the cornea of the squinting eye is similar to that which it formerly occupied on the cornea of the fixing eye. The string keeps the ophthalmoscope hand at 1 meter from the patient's eye. The observer

keeps the tape hand as nearly as possible at the same distance from the patient's eye. The graduated scale on the tape, where it slides through the tape hand, shows approximately the angle of the deviation in degrees."

Various forms of "deviometers" have been devised for measuring strabismus, especially by Nelson Black and by C. Worth.

Treatment of Concomitant (*Comitant*) **Strabismus.**—*A. Convergent Concomitant Strabismus.*

1. *Spectacle Treatment.*—Glasses which neutralize the refractive error should be ordered for every patient with convergent comitant squint after the use of atropin has thoroughly paralyzed the function of the ciliary muscle. In the majority of cases the refraction is hyperopic and is often associated with considerable degrees of astigmatism.

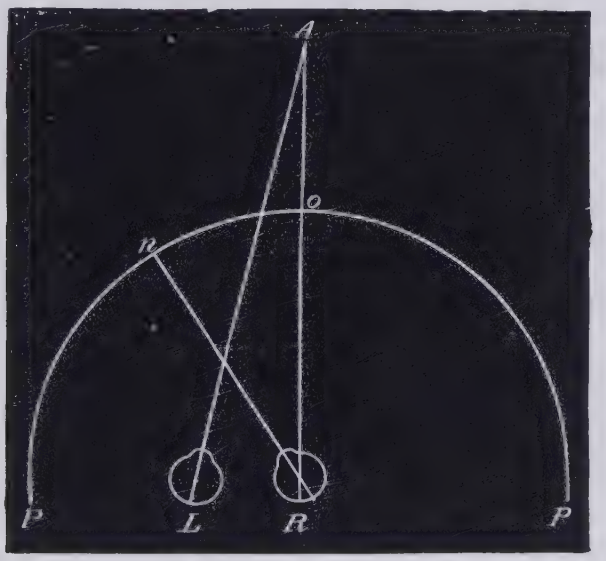


FIG. 273.—Measurement of squint with a perimeter.

There is no difficulty in estimating exactly the proper lenses by means of retinoscopy, and if they are persistently worn early enough—before the fifth year—and, in addition, fusion-training is carried on, the strabismus will be cured in a very considerable percentage (variously estimated from 30 to 70 per cent.) of the cases. It is important that this non-operative treatment of squint should be begun as soon after the discovery of the condition as possible, and glasses may usually be adjusted when the child is three years old; often much earlier.

Prolonged atropinization of both eyes of very young children with squint, in order to remove the abnormal stimulus to convergence which results from overaction of the ciliary muscle, was at one time a much recommended method of treatment. As Worth points out, while it may produce temporary improvement, or even disappearance of the strabismus, it tends to increase the amblyopia of the deviating eye, and

is, therefore, a therapeutic measure to be condemned. He properly recommends atropinization of the fixing eye only, so that the child shall acquire the habit of using the better (atropinized) eye for distant vision, and the poorer (unatropinized) eye for close vision, and thus avoid amblyopia from disuse. Suitable glasses should be worn. If the visual acuteness rises sufficiently, so that the originally deviating eye becomes the squinting eye, the drug must be discontinued, and, if the original condition repeats itself, be used carefully and intermittingly; for example, for a few days during each month (Worth). Reber recommended invisible bifocal lenses in the treatment of esotropia in little children, a sphere of 2 to 3 D being added to the correcting lenses, the idea being "to set the spastic accommodation apparatus at rest." A 0.5 per cent. solution of atropin is used once daily for two months; at the end of this time it is discontinued. A similar recommendation has been made by Linn Emerson. These recommendations are in practical accord with methods previously advocated by Theobald. According to Duane accommodative insufficiency is more often associated with insufficiency of convergence. Rarely, however, in his experience, persistent convergence excess is not relieved by wearing the full correction for distance and near. In such circumstances the "bifocal treatment" may afford relief.

2. *Educative Treatment.*—This includes occlusion of the eye by means of a *shade* or *pad, bar reading, orthoptic training,* and *development of the fusion-sense.*

(*a*) *Occlusion of the Fixing Eye.*—The sound eye should be covered with a shade or bandage, not so much with the hope of improving the acuteness of vision of the deviating eye, but, as Priestley Smith has said, to compel it to use such vision as it has to promote fixation, and to prevent or stop the habit of suppression. If the child wears spectacles, as it should, a blinder of gutta-percha may readily be adjusted on the lens in front of the fixing eye. If the vision of the squinting eye is very imperfect, it is permissible during this treatment to wear the patch on this eye instead of the sound one for a few hours each day; but both eyes should not be allowed to be uncovered at the same time. Occlusion of the fixating eye is a method of real value. Naturally, the earlier it is used and the more persistently it is employed, the better will be the results.

(*b*) *Orthoptic Training.*—This consists of the establishment of diplopia and training the eyes to fuse the double images, and is a method of treatment of squint which was especially advocated by Javal. It is particularly suited to moderate degrees of strabismus and to residual squint after operation. It requires considerable care and patience properly to carry out the details. In order to educate the fusion faculty a *stereoscope* should be employed, the patient's ametropia having been fully corrected. The illumination of the object looked at by the deviating eye may be increased in order to reinforce its visual impressions, as in Landolt's new stereoscope.

If the angle of squint is very great, both eyes cannot look at the

same time into an ordinary stereoscope, and therefore a number of excellent instruments have been devised which can be adapted to the angle of squint. Kroll's orthoptic exercises, arranged by Perlia, which consist of colored plates placed in a suitable stereoscope, are useful. In many respects with the instrument devised by Claud Worth, to which he has given the name *amblyoscope*, the most satisfactory results are achieved, and, as this accomplished surgeon's method is now so much employed, the following directions have been written at the author's request by a member of his staff, Dr. H. Maxwell Langdon, who has devoted much attention to these exercises:

The amblyoscope consists of two tubes, one for either eye, each having its own illumination, which can be increased or lessened so as to equalize the visual impressions in case one eye is amblyopic. An object-slide is placed in the objective end of each tube, and is reflected in a mirror at the bend of the tubes, which is placed at the focal distance of convex lenses fitted in the proximal ends of the tubes, so that no accommodation is necessary. The proximal ends of the tubes are hinged in such a manner that they may be adapted to a convergent strabismus of 60° or a divergent strabismus of 30°.

The child's vision should be tested, if types or other signs cannot be utilized, with small white ivory balls, each with a diameter varying from ½ to 1½ inch. Each eye is tried separately, and the child is required to pick up the ball, which is rolled with a twisting movement. If this test reveals that one eye is amblyopic and possesses one-sixth or less of visual acuteness, some form of blinder exercise should be instituted to improve the defective visual acuteness (see page 632). Preceding the exercises with the amblyoscope, the angle of strabismus should be measured according to the methods elsewhere described, the refractive error having been carefully and fully corrected, and the glasses being in position, and during all of these exercises the glasses must be worn. Amblyoscope training should be begun as soon as the child is old enough to look at ordinary pictures and to talk about them, because deviation yields far more readily to these exercises in young children than in older ones, and, moreover, after the sixth year it is usually practically impossible to make any satisfactory impression upon the defective fusion faculty. A child of three years is well able to take part in these exercises, especially if they are so conducted that they represent to him a game in which he may readily be interested. Usually one or two sittings a week, each occupying half an hour, are sufficient. The child should be seated on a chair between the surgeon's knees, and the angle of the tubes approximated to the angle of strabismus. Next, the illumination, which may consist of two electric-light bulbs, two lamps, or two candles, equally distant from each tube, is arranged, and an object-slide is placed in each holder. These object-slides should consist of pictures familiar to young children, but the ones used at first should be quite dissimilar; for example, the picture of a bird and the picture of a cage. The child is now required to look into the tubes, and is asked what he sees. If one eye is amblyopic to any considerable degree, it is probable that the image of the object before the better or fixing eye will be the only one which is visible. Hence, the illumination must be altered before the other object-slide can be seen by diminishing the light before the fixing or better eye, and increasing that before the amblyopic or squinting eye, continuing with this regulation of lights until both objects are visible and can be described by the child. This alteration in the lights can be accomplished in various ways; for example, by changing the distance of the lights, as Mr. Worth suggests, or by adapting to the amblyoscope, as the writer has done, a revolving wheel, which contains smoked lenses of different densities, and which can be turned before the non-amblyopic eye. Each object should be seen clearly, and the exercise should be varied with several pairs of object-slides. Next, the child is required to place one hand on each of the surgeon's knees and to tap that knee on the side on which the picture

of the bird is seen. If the angle of the tubes is rapidly altered a position will be found where the slightest movement of the tubes causes the picture of the bird to pass directly through from one side of the picture of the cage to the other. But, after continuing the exercises, the bird apparently will go directly into the cage, indicating that the child is acquiring a certain amount of fusing power. If one object is above the other, this vertical deviation must be overcome by means of prisms suitably placed in the grooved slides back of the focusing lenses. Dr. Nelson M. Black has added a vertical adjustment to the Worth amblyoscope,[1] which simplifies the correction of this deviation (Fig. 274). As soon as the child can easily merge the two objects, more difficult tasks are set, with slides demanding accurate and complete fusion, and by gradually widening the angle of the tube a range of fusion which varies from 5° to 15° may be acquired by the patient.

Finally, a series of stereoscopic pictures, intended to teach the child the sense of perspective, are employed. During these exercises, by which the fusion faculty is stimulated and developed, the strabismus may do one of three things: it may



Fig. 274.—The Worth-Black amblyoscope.

disappear after a few days of training; it may gradually lessen; or it may not alter at all, and operative procedures are required to produce parallelism of the visual axes.

These methods to overcome the defective development of the fusion faculty should be faithfully tried in spite of the trouble which their use entails. Certainly the re-establishment of binocular vision in these circumstances is worth every effort.

[1] The author uses, with much satisfaction, Dr. A. Maitland Ramsay's (The Ophthalmoscope, January, 1905) modification of Worth's amblyoscope, in which totally reflecting prisms are employed instead of mirrors. Back of each picture is placed a small electric lamp, the relative brightness of which can be varied to any desired extent by shifting a key, which increases the resistance for one of the lamps while it diminishes it for the other. The author has slightly modified this instrument by adding to it an arrangement by which prisms to correct vertical deviations may be inserted, and scales which indicate the exact separation of the tubes to suit the interpupillary distance and the degree to which the tubes must be converged or diverged, according to the character and angle of the squint.

(*c*) *Bar Reading* (*Controlled Reading of Javal*).—A pencil or, as Priestley Smith suggests, a thin strip of metal is held midway between the eyes and the book which they regard. Reading can then take place without interruption only if both eyes are employed. Priestley Smith describes the exercises as follows: "When the patient's fixing eye reaches that portion of the line which is hidden from it by the bar, he must use his other eye. Then the fixing eye is covered for a moment with a screen. Next, the patient is taught to occlude it for himself by a momentary closure of the lids. Soon he will be able to travel along the line with only a slight hitch where he closes the better eye, and at last he will read smoothly, keeping both eyes open." The method is chiefly effective when practised in conjunction with the use of the shade—that is, the shade covers the fixing eye and it is uncovered only for the purpose of bar reading; and this should be practised as much as possible. Indeed, according to Javal, the exercises must be continued for months, but there seems no doubt that they are efficient aids in the recovery of binocular vision. They are valuable in the residual squints after operation.

3. *Operative treatment* consists of tenotomy of one or both internal recti, with or without advancement of the externi, or of bilateral advancement of the externi without tenotomy of the interni (see page 784). If possible, operation should not be undertaken until the fusion faculty has been developed by the exercises already described, and in no circumstances until the refractive error has been fully corrected and glasses have been worn for at least six months. If, in spite of such treatment, the deviation remains constant, operation is necessary, and may be performed if the child has passed the sixth or seventh year. If the exercises have failed to develop the power of binocular fusion, or if these exercises have begun at a time too late to expect this result, it would seem wise, under most conditions, as E. Jackson insists, to wait until the patient has reached an age when the operation can be performed under local anesthesia and intelligent co-operation secured; to wait, in short, until after the period of rapid growth and development Worth, on the other hand, has no objection to general anesthesia.

There is some difference of opinion in regard to the operations which should be practised for the relief of convergent strabismus, and each case must be carefully studied before a correct decision can be reached. The practice, at one time almost universal, of endeavoring to correct convergent strabismus by means of tenotomy of one or both interni, according to the amount of the deviation, has been largely and very properly abandoned in favor of advancement of the externi.

While it is true that in small squints (15° to 20°) tenotomy of the internus of the deviating eye, if this is not seriously amblyopic, will often yield, temporarily at least, a satisfactory cosmetic result, and that in *alternating squint*, with good vision of each eye, even a double carefully performed tenotomy has been recommended, in most circumstances a tenotomy should be avoided. As Landolt has well said, the "dosage" of tenotomy is uncertain, and from the dynamic stand-

point its effects are unfavorable. The outward rotation tends to increase, the palpebral fissure is widened, the eye often is slightly prominent, and inward rotation is permanently weakened. Free division of the tendinous insertion of the interni and the surrounding capsular attachments is *never permissible*, and almost sure in subsequent years to lead to divergence; indeed, this may be the result of bilateral tenotomies of the interni, even if they are carefully performed. Admirable results follow bilateral advancement of the externi close to the cornea (tenotomy is not performed), and in pronounced squint this may be combined with resection of more or less of the muscle. In this respect the author can confirm the value of Landolt's advice and method, and is in full accord with Claud Worth's condemnation (with few exceptions) of tenotomy. In slight degrees of squint simple advancement will usually suffice. After operation either both eyes should be bandaged until the sutures are removed, or both eyes should be unbandaged and the patient from the first directed to wear his correcting glasses.[1] (For the methods of performing tenotomy and advancement, see page 781.)

B. Divergent Concomitant (Comitant) Strabismus.—The relation of divergence-excess and convergence-insufficiency to divergent strabismus is outlined on page 639. A divergent squint may begin as a periodic defect and be gradually converted into a continuous defect. The treatment of this form of comitant squint includes the correction of the error of refraction with suitable glasses, training convergence, and operative measures.

(*a*) *Glasses* which neutralize the refractive error (most commonly myopia or myopic astigmatism if convergence-insufficiency is the predominating muscular error) should be adjusted according to the rules which are given in the chapter devoted to the measurement of abnormal refraction. H. Landolt recommends concave glasses of such strength that they overcorrect the myopia. According to him the excessive effort of accommodation stimulates convergence. According to Wootton hyperopia is much more commonly associated with divergence-excess than myopia; the reverse is true in convergence-insufficiency where myopia is the rule. In the mixed form, that is, marked divergence-excess and convergence-insufficiency, he finds that anisometropia is frequently present, myopia and hyperopia being less commonly the associated refractive defect. Naturally, as he admits, there are exceptions to these rules. He doubts the value of correcting glasses if divergence-excess is the predominating error. Moderate degrees of divergent deviation may often be favorably influenced by prismatic exercises (see page 643).

(*b*) *Operative measures* depend entirely upon the degree of the deviation, the vision in the diverging eye, and the cause of the difficulty. When true divergent strabismus exists it is usually necessary to per-

[1] For an important paper on this subject the reader is referred to "The Indications for Operating in Heterophoria and Squint," by Alexander Duane, Archives of Ophthalmology, vol. xl, No. 4, p. 390, 1911.

form an operation to correct it. This may be either tenotomy of one or both externi, or this operation may be combined with advancement of the internal rectus. Advancement of the interni is preferable to tenotomy of the externi if there is convergence-insufficiency. Should the condition be one of divergence-excess with hyperopia, tenotomy of the externi is indicated; indeed, this also obtains if myopia is present. A coexisting vertical deviation should be remedied, and some operators (Hansell and Reber) prefer to make the vertical adjustment before attacking the lateral deviation. The procedure in some cases abolishes the lateral squint. Divergent strabismus due to overcorrection following tenotomy of the interni should be remedied by advancement of the severed internal recti muscles.

Vertical Strabismus.—Often there is an *apparent* vertical deviation associated with lateral strabismus, which disappears with the correction of the latter defect. In cases of *true* vertical deviation requiring operation Worth recommends advancement of the inferior rectus muscle of the eye which turns upward. Landolt, although opposed to tenotomy of the lateral muscles, does not object to this operation on the superior rectus. All surgeons agree that tenotomy of the inferior rectus usually is not advisable, except as it may be performed to compensate for the diplopia occasioned by a paralysis of the superior oblique of the opposite eye. In certain circumstances an upward deviation is due to spasmodic action of the inferior oblique and must be corrected by tenotomy of this muscle (see page 783).

Results of Operation in Convergent Strabismus.—The effect of the operation, if well performed, is to produce parallel visual axes, and thus remove the disfigurement. Properly speaking, a *cure* is obtained only when there is improvement in the vision of the squinting eye and binocular vision is secured. There has been much difference of opinion on this subject, and some authors—for example, Lang and Barrett—have questioned if valuable improvement in the vision of the amblyopic eye ever takes place. Binocular vision is secured in a small number of the cases, after correctly performed operations and after the patients' eyes have been carefully corrected with glasses and trained by orthoptic exercises. It must be remembered, as Duane points out, that a patient whose squint has been "cured" may have binocular fixation, but monocular vision, that is, suppression of one image continues. Careful training of the fusion faculty should begin early and the exercises already described should be systematically carried out. Indeed, as may have been inferred, the necessity for operation, if only the educative treatment of strabismus is begun soon enough, is sure to diminish, but should an operation become necessary, it is also sure to be followed by far better results than can be achieved in the absence of such training.

It is often difficult to ascertain whether true binocular vision exists, especially in young children, and successful bar reading, usually quoted as a sufficient test, is, according to Priestley Smith, not without its fallacies. This author tests as follows:

A reversible frame carrying red and blue glasses is placed in front of the patient's spectacles, and he is shown, at the reading distance, a card with three disks on a black ground, a white one in the middle, a red one above, and a blue one below. If he can see all three at once and in a line, he is probably using both eyes and fusing the two images of the white disk. If with each eye alone he sees two, but with both eyes three, the proof is fairly positive. The test may be improved by placing a black letter on each disk, which, if the patient has sufficient vision, he should read. The same test with larger objects may be used at longer ranges. The light should be good, but not too strong, and not artificial. Hering's drop-test may also be employed. A simple and ingenious diaphragm test for binocular vision has been devised by N. Bishop Harman.[1]

Spastic Strabismus.—This condition, more properly characterized by the term *convergence cramp*, or *spasm* (*non-accommodative convergence excess*) is seen in hysteria, and is characterized by convergent squint, limitation of the motility of the external recti, and by homonymous diplopia. It somewhat resembles paralysis of the abducens, for which it may be mistaken, but from which it should be differentiated by a study of the double images. Sometimes this form of strabismus or, rather, convergence spasm is associated with other hysteric manifestations—blepharospasm and ptosis—and may be a symptom of meningitis. Sometimes what appears to be a palsy of the external rectus in patients the subjects of a psychosis is really a convergence-spasm, as may be proved by instilling a solution of atropin (Duane).

Abnormal Balance of the Ocular Muscles, or Heterophoria (*Latent Deviation*).—This is, as already defined, a disturbance of the normal balance of the exterior eye muscles, which creates a tendency for the visual lines to depart from parallelism, a tendency which is checked by the habitual desire for binocular vision, or that vision in which the images of an object formed on the retinas of the two eyes make but a single mental impression.

Heterophoria (*imbalance*, according to Gould) differs from *squint* or *heterotropia* because in the latter the fusion of the images is usually impossible—*i. e.*, binocular single vision is absent—and there is an evident departure of the visual lines from parallelism, which gives rise to the term which designates the condition.

Causes.—Imbalance of the ocular muscles may be due to: (*a*) weakness of the muscles (properly called insufficiency) of congenital origin, or depending upon a general lack in muscular tone, the result of anemia, nervous exhaustion, pelvic disorders, etc., or malaria, rheumatism, gout, etc., diseases which, however, may also be potent by affecting not the muscles themselves, but their innervation; (*b*) errors of refraction and disturbance of accommodative efforts (*accommodative heterophoria*); (*c*) the anatomic arrangement of the parts—for example, faulty attachment of the muscle (*comitant heterophoria*); (*d*) excessive

[1] For description, see Ophthalmic Review, vol. xxviii, 1909, p. 93.

action or spasm of opposing and dominating muscles (*spasmodic heterophoria*); (*e*) disturbances of innervation (*central heterophoria*); and (*f*) a paretic condition of the muscle (*paretic heterophoria* of Duane).

Varieties.—According to Stevens' nomenclature, if there is a tending of the visual lines in parallelism, the term *orthophoria* is applied; if there is a tending of these lines in some other direction, the term *heterophoria* is employed. Heterophoria is divided into: *esophoria*, a tending of the visual lines inward; *exophoria*, a tending of the visual lines outward; *hyperphoria* (right or left), a tending of the right or left visual line in a direction above its fellow. *Cyclophoria*, according to Savage, is a want of equilibrium on the part of the oblique muscles (see also page 81).

Abnormal inward tending of the visual lines may depend upon excessive convergence or deficient divergence, or upon these conditions combined. Duane[1] describes the signs as follows: If *esophoria* for distance is less than for near, abduction (prism-divergence) not disproportionately low, adduction (prism-convergence) readily performed, esophoria marked at the near point and the convergence near point excessive, *convergence-excess* is present. If esophoria for distance is much greater than for near, abduction (prism-divergence) disproportionately low or absent, adduction (prism-convergence) normal or subnormal, esophoria slight, absent, or replaced by exophoria at the near point, and the convergence near point not abnormally close to the nose, *divergence-insufficiency* is present.

Convergence-excess is followed, if of long standing, by divergence-insufficiency, and similarly divergence-insufficiency by convergence-excess. In the *mixed form* thus produced there are marked esophoria for near and far, excessive approximation of the convergence near point, and limited, absent, or negative abduction (prism-divergence). Finally, the deviation ceases to be latent, binocular vision is lost, and *esophoria* passes into *esotropia*.

Abnormal outward tending of the visual lines may depend upon deficient convergence or excessive divergence, or upon these conditions combined. Duane records the symptoms as follows: If *exophoria* for distance is slight or absent, abduction (prism-divergence) not very great or even subnormal, adduction (prism-convergence) exceedingly difficult, exophoria marked at the near point, and the convergence near point less than 3 inches and maintained only for a moment, there is *convergence-insufficiency*. Sometimes this may be so great as to constitute a *convergence-paralysis*. If exophoria for distance is marked, abduction (prism-divergence) is high, adduction (prism-convergence) normal or not greatly subnormal, and the convergence near point normal, there is *divergence-excess*.

Convergence-insufficiency is followed, if of long standing, by divergence-excess, and, similarly, divergence-excess by convergence-insuffi-

[1] American Text-book of Diseases of the Eye, Ear, Nose, and Throat, edited by de Schweinitz and Randall, page 515. The descriptions which follow are condensed from Duane's article.

ciency. In the *mixed form* thus produced there are marked exophoria for near and far, excessive abduction (prism-divergence), and marked retreat of the convergence near point. Finally, the deviation ceases to be latent, binocular vision is abandoned, and *exophoria* passes into *exotropia*.

If hyperphoria varies noticeably in different directions of the gaze, it is *non-comitant* and is due to underaction or overaction of one or more of the elevators or depressors; it may be due to spasmodic action of these muscles and may spontaneously disappear. If hyperphoria remains the same in all directions of the gaze, it is *comitant*, and may be due to excessive sursumvergence, or more frequently to the same agencies which produce non-comitant hyperphoria which has become comitant.

Whether the hyperphoria is due to overaction or underaction of one or other set of muscles may be determined by examining the rotations of the eyes (see page 603). Excessive upward rotation would naturally indicate overaction of the elevators of the hyperphoric eye, and excessive downward rotation overaction of the depressors. Deficient upward or downward rotation would indicate underaction of the vertical muscles, and in these circumstances diplopia is readily elicited, as it is in paretic condition, by carrying the test-light in the direction of the action of the affected muscles. Hyperphoria usually does not tend to increase, and therefore binocular fixation is usually retained, and it is comparatively rare for *hyperphoria* to pass into *hypertropia*.

Full correction of hyperopia disturbs the relative range of accommodation and convergence and may cause exophoria (convergence-insufficiency—relative insufficiency of the interni, according to Risley). The same condition is seen in myopes who do not use glasses at close ranges and in presbyopes whose reading-glasses are too strong. Suitable glasses, or a modification of the glasses, and sometimes exercises with prisms, will relieve the condition (see also page 163).

Relative Frequency of Heterophoria.—Faulty directing power of the vertical muscles (hyperphoria) is usually stated to be the least common of these anomalies, but is much more frequent than was once supposed, and, according to Hansell and Reber, will be found in one-third of the cases of refractive anomalies. Many of these hyperphorias, however, are temporary in character and require no treatment except correction of the refraction and any underlying constitutional condition. The power of hyperphoria in causing asthenopic symptoms is of paramount importance, and, according to Stevens, its rôle in disturbing the action of the lateral muscles is significant. As measured at the distant point esophoria is more frequently recorded than exophoria.

Difference Between Heterophoria and Heterotropia (Squint).—The essential difference between these two conditions has already been several times defined, and the passage of a heterophoria into a heterotropia has been described. The differential diagnosis should depend upon the results obtained from the application of certain tests. Duane describes these as follows:

"If there is any noticeable deflection behind the screen (see page 76), the *screen-test is applied in a second way* or by *binocular uncovering*. This procedure consists in covering the left eye and then uncovering both eyes and noticing the movement that takes place. If, on thus uncovering the left eye, the right eye remains steady and the left moves into position, the patient has binocular fixation, and the deflection was a heterophoria and not a squint. If, however, the right eye should move out of its position and the left eye should move into place, there is a squint and the left is the fixing eye. If neither eye moves, there is a squint and the right is the fixing eye. By repeating this experiment with each eye alternately the examiner can tell whether there is a habitual binocular fixation, an alternating fixation, or a monocular squint. The diagnosis between the three may be conveniently formulated as follows:

"1. If in binocular uncovering *but one eye moves*, heterophoria and not squint exists.

"2. If either *both eyes move* or, in spite of there being an evident deviation, *both eyes remain steady*, squint exists.

"3. In the latter case if, when the left eye is uncovered, the eyes behave in the same way as they do when the right eye is uncovered (both alike moving or both alike remaining steady, no matter which eye is uncovered), the squint is *alternating*.

"4. If when one eye—for instance, the right—is uncovered, both eyes move, and when the other eye (in this case the left) is uncovered both eyes remain steady, the squint is *monocular* (confined in this case to the left eye)."

Symptoms.—These are usually classified under the general term *muscular asthenopia*, and may be divided into the *ocular* and the *general* symptoms.

To the *first group* belong pain, often over the insertion of the affected muscle, and especially marked when the eye is suddenly moved in the direction of its action; blurred vision and imperfect power of working at close ranges; inability to gaze attentively at a stationary object even at long ranges, and great discomfort when attempting to watch moving objects; dread of light and blepharospasm, often confined to a few fibers of the orbicularis; and local congestions of the conjunctiva, especially over the insertion of the muscle, and on the margins of the lids. Often there are eccentric poses of the head, distortions of the features, especially wrinkling of the forehead, contractions of the sternocleidomastoid, and tilting of one or other shoulder.

In the *second group* the prominent symptom is *headache*, which may be situated in any portion of the cranium, but which is common in the occiput. The pain may immediately follow the use of the eyes, or be delayed, or come on at a certain hour of the day, or even night. The headache may assume the migrainous type.

Pain in the back, especially between the shoulder-blades, or precordial pain, is common. Vertigo, generally subjective, is frequent, one variety being characterized by a sense of falling forward when walking

in a crowd, associated with confusion of ideas. Drowsiness and, on the other hand, insomnia may be present, and a variety of general or so-called reflex neuroses.

Chorea, epilepsy, pseudochorea, night-terrors, melancholia, neurasthenia, hysteria, palpitation of the heart, indigestion, constipation, flatulent dyspepsia, and a host of other complaints have been attributed to muscular imbalance, and also to accommodative strain, and under these conditions the eyes should always be examined and the ocular defects corrected (see also page 154). Many instances of remarkable nervous disturbances are associated with heterophoria, especially hyperphoria (as well as with refractive error), and cure will often follow the relief of the ocular difficulty. Unfortunately, the whole matter has not always escaped exaggeration.

Method of Examination.—The method of examining the ocular muscles has been fully described on pages 76–83. (See also Appendix, page 801.) Two points deserve reiteration—viz., that a measurement of the relative weakness and power of the muscles is inexact unless this has been made after the refractive error has been corrected, and the muscles have been tested through the correcting lenses; and that the examinations of the muscles should be made both for the *near* and the *far* point—*i. e.*, at 30 cm. and 6 meters—the latter being the more important determination.

Treatment.—As Duane has well said, "There must be no attempt to treat an insufficiency simply as an insufficiency, but account must be taken of the complex causes which lie at the root of it."

Strict orthophoria is rare. Small errors of the lateral muscles are often unimportant.

If there is a constitutional disorder or an insufficient nervous tone, this must be treated on general principles. Strychnin or ascending doses of tincture of nux vomica have been recommended, and for the purpose of improving the general tone of the system should be administered. Galvanism may be tried, but it is doubtful if the current reaches the muscle. Large doses of tincture of hyoscyamus are of distinct advantage in cases of spasmodic heterophoria.

In every case of heterophoria the refractive error should be corrected according to the rules already laid down. In many instances this alone will suffice to restore the balance and cure the asthenopia. In esophoria of accommodative origin the total amount of the hyperopia and astigmatism should be neutralized with suitable glasses, which are to be worn constantly, and in high degrees of this muscle defect a convex sphere of from 1 to 2 D may be added to the distance correction and used in near work in order to diminish accommodative overactivity (see also page 137); in exophoria, especially with insufficiency of convergence, the full correction of the myopia should be ordered (compare with page 147). Where esophoria exists with myopia and exophoria with hyperopia, this plan must be modified, and an undercorrection of the refractive error prescribed. Convergence-insufficiency caused by glasses of improper strength has been described on page 640.

If the symptoms continue, recourse should be had to *gymnastic exercises with prisms.* The object is to strengthen abduction and adduction. A number of methods are in common use:

1. The patient is instructed to practice fusing the double images produced by viewing a candle-flame or small circle of electric light situated 6 meters away. Abduction (prism-divergence) and adduction (prism-convergence) are exercised, beginning with the weakest prisms and gradually increasing to the strongest. This plan probably acts, as Maddox suggests, by training the efforts of accommodation and convergence to assume broader relations to each other in their work. It is efficient in selected cases.

2. Rhythmic exercises, contraction and relaxation of the muscle being secured by causing the patient to view a test-light 20 feet distant through *adverse prisms,*[1] which are lowered and raised at regular intervals of five seconds, beginning with weak and gradually going to stronger numbers. This is the method of Dr. G. C. Savage. This author also recommends rhythmic exercises by rotating convex cylinders before the eye for the relief of *cyclophoria.*

3. The patient is provided with prisms double the primary distant adduction power. The candle-flame is then slowly carried, while he regards it fixedly and continuously from the near point to the distant point. This is repeated until, without difficulty, he can, through the prisms, secure a single image in all parts of the room. The strength of the "handicap-prisms" should be gradually increased. For esophoria the reverse of the plan is pursued. This is the method which was advocated by Gould.

Referring to muscle exercises, Duane states that he regularly employs four, namely: distant exercise with prisms, bases out; exercise with prisms, bases out, at near points; exercise with prisms, bases in, at near points, and exercises in converging on a pencil-point (see also page 78). Exercises with prisms, bases out, are followed by most satisfactory results in exophoria, especially in convergence-insufficiency, and should always be practised not only at the distance, but, as Duane insists, at the near point. Exercises with prisms, bases in, in esophoria at the distant point, in the author's experience, have not been of any value, but recent experience, based on Duane's advice to use diverging prisms at near points in cases of convergence-excess, indicates that the method may produce good results. The author has failed to observe relief in hyperphoria from prismatic exercises, but Savage's method has received the commendation of many competent observers, and should be tried.

The next method of treatment is the *prescription of prisms.* The action of prisms has been explained (see page 18). Much difference of opinion exists in regard to their therapeutic value. The author believes with Duane that "the employment of prisms in lateral deviations

[1] "Adverse prism" is a term used by Maddox, and means one with its apex set in the opposite direction from a "relieving prism," for example, base out if the interni are to be affected, base in if the externi are to be exercised.

is to be avoided except as a temporary measure, since prisms, base in, tend to produce convergence-insufficiency, and prisms, base out, convergence-excess, so that in both cases they ultimately increase the deviation which they are designed to correct." Prisms may be ordered when the range of movement is perfect but in an unavailable position. The base of the prism should be placed toward the muscle which is to be aided, and the apex toward the muscle which is to be weakened.

It is usually uncomfortable for the patient to wear more than 4° or 5° constantly—*i. e.*, 2 or $2\frac{1}{2}$ over each eye. This statement, however, admits of many modifications, and often the strength of the prism may be increased much beyond this limit.

In permanent latent deviations of the vertical muscles (right or left hyperphoria) the defect is often quite small, and usually not above 4° or 5°; hence prisms may readily be ordered for continuous use, and combined with the lenses which correct the refractive error, forming a *prismosphere*. If, for example, there is right hyperphoria of 2°, a 2° prism base down before the right eye corrects the difficulty, or, what is equivalent, the prism may be divided between the two eyes—*i. e.*, 1° base down before the right, and 1° base up before the left. It is safe to correct very trifling errors in the vertical muscles either with prisms or by decentering the correcting lens to an equivalent degree (see page 20), providing these errors are still maintained after continuous use of glasses which neutralize the refractive error.

In esophoria, which is a frequent cause of muscular asthenopia, prisms are often combined with the correcting lenses and worn constantly. For the reasons before stated the author doubts the value of constant prisms, with rare exceptions, under these conditions.

In exophoria the constant use of prisms is not advisable. On the other hand, they may be a great help in relieving the strain upon convergence by removing the point of intersection of the visual axes farther from the eyes, and for this purpose they are combined with reading glasses. In high degrees of exophoria, or if there is actual divergence, abductive prisms are of little use; if the deficiency of the directing power is determined to be equivalent to 10°, one-half of this may be corrected—*i. e.*, $2\frac{1}{2}$° base in over each eye; if it is desired to remove all effort, the faulty tendency is measured in the usual way, and if it is within suitable limits, prisms are ordered, combined with the correcting glasses which neutralize the defect.[1]

It has also been suggested to strengthen the muscles by means of *orthoptic exercises*—*i. e.*, by causing them to make forced movement in different directions; by making forced movements of convergence, the patient being required to look at near objects—"thumb exercises"; by requiring the eyes to unite the images of two slightly separated objects. *Stereoscopic exercises* are also of advantage.

[1] When a spheric lens is combined with a prism, the deviating effect of the combination is different from that of the prism alone. Mr. Archibald Percival (Ophthalmic Review, October, 1891) has constructed elaborate tables which give the deviating effect.

In the event of failure to relieve asthenopic symptoms by the methods thus far described operative procedure may be necessary. This consists of partial, complete, or graduated tenotomy of the antagonistic muscle, or of advancement of the feeble muscle (see chapter on Operations). Whether advancement or tenotomy should be performed depends upon the conditions. Advancement is indicated to strengthen a weak muscle, and tenotomy if an overstrong muscle is to be weakened. For example, in exophoria due to convergence-insufficiency advancement of the internus is a more rational procedure than tenotomy of the externus, but if the exophoria depends upon divergence-excess, tenotomy of the externus is the better operation. The same advice applies to esophoria, convergence-excess indicating tenotomy of the internus, and divergence-insufficiency advancement of the externus. According to circumstances, one or both externi or interni may need readjustment or division. Operations on the vertical muscles must be governed by similar rules. Surgical interference is required only after all other measures have been long and faithfully tried and have failed to give relief. While cases of muscular imbalance, best treated by operative interference, are encountered (aptly called by Risley "absolute insufficiencies," equivalent to the structural and insertional anomalies of Duane), in the opinion of the author they represent a limited proportion of the whole number. Moreover, as our knowledge of the etiology of abnormalities of muscular balance increases and our methods of non-surgical treatment improve, this number grows steadily smaller.

So-called graduated tenotomies and partial tenotomies are performed by some surgeons, and it is asserted that adjustments are exactly made, but in them the author has little faith. It is true that brilliant results have been achieved and described by experienced operators, but there is no doubt that a good deal of injudicious "snipping of the tendons of the ocular muscles" has been practised.

Nystagmus.—This term is applied to a condition characterized by an involuntary, rapid movement of the eyeballs. The movement may be lateral, vertical, rotary, or mixed—*i. e.*, a compound of two varieties. According to the character of the movement there are two chief types of nystagmus, namely, the *undulatory form* (also called *vibratory nystagmus*) in which the movements have the same to-and-fro velocity, and the *rhythmic* form (also called *resilient nystagmus*) in which a comparatively slow movement (slow phase) in one direction is followed by a rapid return movement in the opposite direction.

The condition may be *congenital* (probably begins in very early life) or *acquired*, and is bilateral in the majority of cases, although a few instances of unilateral nystagmus have been reported, with the movements usually in the vertical direction. It is possible, however, inasmuch as slight forms of nystagmus are detected only by using the ophthalmoscope and watching the fundus, that some of these supposed unilateral cases have actually been bilateral.

Congenital nystagmus is associated with cases of defective construc-

tion of the eyeball—coloboma, microphthalmos, etc. It is also common in albinism and in color-blind persons with small central scotomas (C. L. Franklyn). Nystagmus may be due to opacities of the media, especially when such obstruction to the rays of light has been caused by diseases occurring early in life and in blind eyes (congenital cataract, leukoma after ophthalmia neonatorum), to chorioretinitis, pigmentary degeneration of the retina, etc. In blind eyes the so-called *searching movements* occur; that is, the eyes make a comparatively slow and wide movement from the primary position, to which after a time they again return, and so the movements are repeated. *Pseudonystagmus*, usually bilateral, is the term applied to jerky movements which are seen when one or both eyes are rotated near to the limit of their excursion in one or other direction; that is, between this point and the exterior limit of rotation these jerky movements develop. It probably depends upon fatigue of the muscles. It can be developed in various nervous diseases, especially Friedreich's ataxia and multiple sclerosis.

Hereditary nystagmus, extending through a number of generations, has been especially studied by Nettleship: two types exist, the first is male-limited, the transmission being through unaffected females; in the second both sexes are affected, the descent being most frequent through the females. In this class head movement is common.

Nystagmus may be *acquired* in the pursuit of certain occupations, especially mining, and is commonly known as *miners' nystagmus*. It generally occurs among those who use a dim light, and whose work necessitates keeping the eyes in an unusual position for many hours together (Snell). T. L. Llewellyn believes that the chief cause of this form of nystagmus is strain caused by deficient light. Errors of refraction increase the liability to nystagmus. Miners' nystagmus has also been attributed to muscle fatigue and to insufficiency of the fusion power (Dransart and Van Houtte). The visual fields, according to Cridland, are similar to those observed in traumatic neurasthenia.

Nystagmus is common in diseases of the nervous system, particularly disseminated sclerosis and Friedreich's ataxia, as previously noted, but a *true* nystagmus also occurs in these affections. Nystagmus is associated with many diseases of the brain, and has been noted with great frequency in tumors of the cerebellum. Head-jerkings and nystagmus may occur in young children, constituting the so-called *spasmus nutans*. It may be unilateral. *Voluntary nystagmus* has been reported and a rare variety is the so-called *latent nystagmus*, which can be developed only by excluding one eye from binocular vision (Fromaget, Van der Hoeve). The subjects of nystagmus may be greatly disturbed, but not in all its varieties, by the apparent movement of objects, by difficulty in reading and by vertigo; poor vision is common; photophobia may be present.

Nystagmus has been ascribed to chronic fatigue of the muscles and oscillation of the globe consequent upon the muscular atony, and also to a central origin. Duane believes that true nystagmus depends upon

a perversion of the centers for parallel and parallel-rotatory movements and not on peripheral muscle or nerve lesions.

Vestibular nystagmus, produced by irritation of the labyrinth, may be horizontal, vertical, and rotary, and the movement consists of a slow followed by a rapid oscillation, most intense when the visual axes are turned in the direction of the rapid movement, diminished when turned in the opposite direction. This type of nystagmus, an interpretation of vestibular disturbance, can be readily induced by Bárányn caloric test (syringing the ear with hot or cold water), by the rotation test (the rotations being made with the aid of Bárány's chair or one of its modifications), or by galvanic stimulation. These tests have assumed great importance from the otoneurologic standpoint and are of unusual value in examining candidates for service in the aviatio's corps; they have added an important chapter on the localization of intracranial lesions.[1]

Treatment.—If practicable, in cases of nystagmus where there is interference with the reception of perfect retinal images, the best possible vision should be restored by correction of refractive error, by tenotomy, or by iridectomy for new pupil, according to the indications. Very often good results have been noted. If nystagmus is brought about by any occupation, the evident indication is to remove the patient from his surroundings. For central nystagmus from brain or cord disease there is practically no remedy. In some instances of acquired nystagmus benefit has been reported from the local use of eserin and the internal administration of strychnin.

Monocular Diplopia.—This character of diplopia is caused by one of several conditions: By anomalies of refraction, particularly astigmatism; by opacities in the cornea or lens, by subluxation of the lens and by anomalies of the pupil, for example, polycoria (see also page 334); by iridodialysis (page 357); by irregular cramp of the ciliary muscle; by complete or partial constriction of the eyelids, by which they are made to impinge on the cornea (G. J. Bull); by hysteria or allied functional nervous disturbance; by organic disease of the brain or its membranes, associated with abducens paralysis (Gunn and Anderson); by simulation, the symptom being an invention of the patient for the purpose of magnifying the result of injuries.

[1] For a full consideration of nystagmus in these relationships consult "Equilibrium and Vertigo," by Isaac H. Jones, and its chapter containing an analysis of pathologic cases by Lewis Fisher.

CHAPTER XIX

DISEASES OF THE LACRIMAL APPARATUS

Diseases of the lacrimal structure naturally divide themselves into those which have their seat in the lacrimal glands and those which affect the drainage system—*i. e.*, the puncta, canaliculi, lacrimal sac, and nasal duct.

Dacryo-adenitis.—This is an inflammation of the lacrimal gland, a comparatively rare affection, which may be *acute* or *chronic*, *suppurative* or *non-suppurative*.

Non-suppurative dacryo-adenitis, on account of its analogy to bilateral parotitis, has been called *mumps of the lacrimal gland* (Hirschberg). It may be caused by influenza, smallpox, measles, scarlet fever, leukemia, and mumps. *Tuberculous dacryo-adenitis* is rare, according to Stieren, who reports an example of this affection, only 12 cases being on record. The unilateral chronic form of inflammation of the lacrimal gland is more common than the bilateral variety, and has been observed in scrofulous subjects, and may be caused by an injury or follow diseases of the conjunctiva and cornea.

If the gland is chronically enlarged, palpation will reveal its lobulated border; if the inflammation is acute, there are pain, tenderness, and swelling at the upper and outer part of the eyelid, with chemosis of the conjunctiva; the rotation of the eye upward and outward may be limited. This may go on to suppuration, and the abscess usually points upon the skin, but also through the conjunctiva; streptococci, staphylococci, and pneumococci are found in the purulent material. Acute dacryo-adenitis may result from infections from the conjunctiva, from infectious diseases, and from injury. *Metastatic dacryo-adenitis* in the subjects of gonorrhea has been described.

Treatment.—Pads of gauze steeped in hot boric acid lotion or a 40 per cent. solution of sulphate of magnesia may be applied to relieve pain, and at the first appearance of pus an incision should be made either through the integument parallel to the eyebrow or through the conjunctiva. If induration of the gland occurs, this should be treated locally with iodin or iodid of cadmium ointment. A tuberculous gland should be removed.

Hypertrophy of the lacrimal gland has been observed at birth, but usually is seen in later years, and consists in an indurated lobulated tumor having its situation in the upper and outer part of the orbit.

Atrophy of the lacrimal gland, as the result of xerophthalmos, has been described.

Spontaneous prolapse of the lacrimal gland appears in the form of a soft movable tumor under the upper eyelid. Hypertrophy

and prolapse or prominence of the palpebral portion of the lacrimal gland may occur in various corneal and conjunctival inflammations, and is evident on everting the upper lid.

The **treatment** consists of extirpation of the prolapsed organ.

Traumatic Dislocation of the Lacrimal Gland.—This is a rare accident, and occurs most frequently in young children (Villard), but also in adults (E. Jackson). Usually the gland prolapses through a wound in the upper lid. If the gland can be returned to its place and the wound sutured, this procedure is preferable to excision, which, however, may be necessary, especially if infection occurs.

Fistula of the Lacrimal Gland.—This may remain on account of the rupture of an abscess, but has also been recorded as a congenital defect at the outer third of the upper lid. A fringe of hair may surround the opening of the fistula.

The fistula may be closed by repeated cauterization or by a plastic operation; in the event of the failure of these measures, extirpation of the gland is indicated.

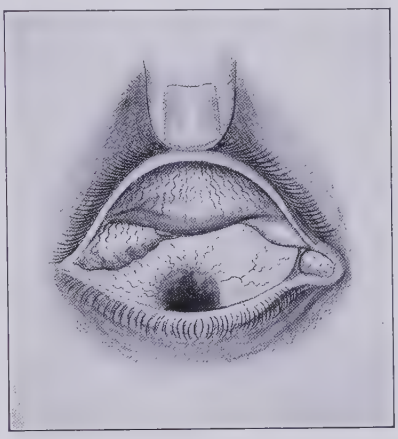


FIG. 275.—Enlargement and prolapse of the palpebral portion of the lacrimal gland in an eye with kerato-iritis.

Syphilis of the Lacrimal Gland.—The lacrimal gland is singularly free from syphilitic affections, but specific induration and inflammation have been described, that is, *syphilitic dacryoadenitis*, in most instances (and there are only a few on record) a gummatous process. A swelling (inflammation) of the lacrimal gland in association with a chancre of the upper retrotarsal fold has been observed (de Lapersonne). The usual antisyphilitic treatment is required. Symmetric enlargement of the glands entirely dissipated by arsphenamin has been reported (H. W. Cowper).

Dacryops.—This affection, often classified with diseases of the conjunctiva, is caused by a cystic distention of one of the main gland-ducts or of one of those of the accessory lacrimal gland, and appears in the form of a bluish, translucent swelling beneath the conjunctiva at its upper and outer part. If the mouth of the excretory duct is not occluded, pressure upon the tumor causes a few drops of liquid to escape. It is a comparatively infrequent affection, about 35 cases being on record (Ernest Thomson).

Tumors of the Lacrimal Gland.—Adenoma, fibroma, myxoma, adeno-angioma, epithelioma, carcinoma, osteochrondroma, lymphoma, cylindroma, and sarcoma occur. *Tubercle*, in the form of a small almond-shaped tumor, has also been reported in this region either in association with systemic tuberculosis or as an isolated lesion. Cysts

and concretions (*dacryoliths*) occur. The concretions are contained in the excretory ducts, and are composed of concentric, chalky masses (Levi). According to Warthin, the majority of lacrimal tumors are, most probably, mixed tumors of endothelial origin, similar to those of the parotid and submaxillary glands. They tend to form cartilaginous, hyaline, and myxomatous tissue, and their malignancy is relatively slight. F. H. Verhoeff believes, however, that mixed tumors of the lacrimal gland are essentially epiblastic in origin, that they are dangerous to sight and to life, and that they should be extirpated as soon as possible, and Laura Lane's statistics (95 cases) show a death-rate of 12.63 per cent.

To a symmetric enlargement of the lacrimal, the parotid, and salivary glands the name *Mikulicz's disease* is applied. Four types have been described (Laura Lane): simple enlargement of the lacrimal and salivary glands; a similar enlargement and moderate tumefaction of the lymphatics (pseudoleukemia) and involvement of all lymphatics and the liver and spleen (true leukemia, a fatal form). According to S. Lewis Ziegler, the tumefactions should be regarded as true lymphomas. The affection has also been ascribed to tuberculosis and to a chronic infection. In the treatment iodid of potassium has been tried, but the drug which has achieved the best success is arsenic. Radiation (*x*-rays) has been recommended.

Anomalies of the Puncta Lacrimalia and Canaliculi.—1. **Congenital Anomalies.**—Double puncta lacrimalia and canaliculi have been observed as *congenital anomalies*, and Majewski has observed quadruple puncta. There may be congenital absence of these structures, or the lacrimal points may be wanting and the canals may be represented by furrows along the edge of the lid—*congenital atresia*.

2. **Acquired Anomalies.**—The slightest change in the natural relation of the lower punctum to the eye, against which it is directed backward, causes *epiphora*, or an overflow of tears.

The most fruitful sources of such abnormal relationship are the various chronic inflammations of the lid and conjunctiva—blepharitis, trachoma, and ectropion—and facial palsy and wounds of this region. In facial palsy, watering of the eye is sometimes an early symptom, and is caused partly by the loss of the compressing power of the lid, especially in the fibers of Horner's muscle, and partly by the falling away of the punctum. An overflow of tears may follow an abnormal position or enlargement of the caruncle. All these conditions cause a *malposition* of the *punctum lacrimale*.

Epiphora is also caused by a stye or tumor of the lid near the punctum, or, if the canaliculus is closed, by the presence of a foreign body, usually a cilium; by a mass of fungus (*streptothrix*), which, by becoming calcified, may form a so-called *tear-stone*, *dacryolith*, and by a *polyp*. Concretions in this region due to the *sporotrichon* have been observed in cases of sporotrichosis of the conjunctiva (Bedell). In like manner chronic conjunctivitis and marginal blepharitis may close either the lacrimal point or the canaliculus. These affections are included under

the terms *stenosis* of the *punctum lacrimale* and *obstruction* of the *canaliculus* (for additional causes of epiphora, see pages 671, 676).

Precisely the opposite condition, namely, *absence of the lacrimal secretion*, has been referred to as a symptom of xerophthalmos (page 254); it may be due to sclerosis or atrophy of the lacrimal gland, for example, in Mikulicz's disease, and has been observed in some cases of facial and trigeminal palsy.

Treatment.—If a foreign body is present, it is usually necessary to slit the canaliculus in order to remove it.

In many cases of epiphora which depend simply upon closure of the lacrimal point this may be opened by means of a gold or silver pin or a *dilator*, which is pushed along the canaliculus. Afterward the permeability of the lacrimal duct may be tested by inserting the point of an Anel syringe and injecting boric acid solution and observing whether it passes freely into the nose. This very simple procedure will often afford great relief without the necessity of either slitting the canaliculus or dilating the duct. If the epiphora has been caused by facial palsy, the treatment advised does not apply. Streptothrix infection of the lacrimal canaliculus is sometimes mistaken for dacryocystitis of the sac and stricture of the duct, because a drop of viscid semipurulent material may be pressed through the puncta. Foreign material in the canaliculus should be carefully examined.

Anomalies of the Lacrimal Sac and Nasal Duct.—1. **Dacryocystitis.**—The symptom in affections of the lacrimal sac and nasal duct which is always present is epiphora; the eye swims in tears, and these are excited to overflow by exposure to dust, cold, or wind; the caruncle and plica are swollen; the neighboring conjunctiva is hyperemic and injected (*lacrimal conjunctivitis*); the skin is macerated, and the margins of the lid, especially toward the nose, show signs of blepharitis.

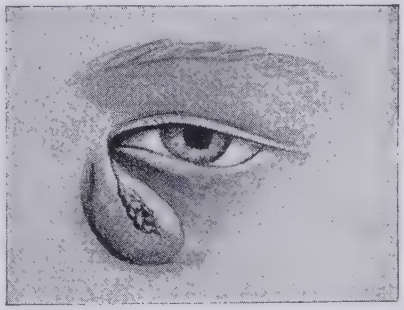


Fig. 276.—Phlegmonous dacryocystitis; pouting granulations surround the fistulous orifice (from a patient in the Children's Hospital).

Usually there is slight distention over the region of the lacrimal sac (*mucocele, lacrimal tumor*), and pressure upon this expresses through the puncta the retained fluid, which is a clear or semitransparent viscid mucus (*dacryocystitis catarrhalis*), or turbid from mixture with purulent material (*dacryocystitis blennorrhoica*).

This *chronic* distention of the lacrimal sac is liable to develop into a suppurative inflammation producing *acute dacryocystitis*, which may be preceded by fever and chill; the lids and region of the nose become tense and tender to the touch, and a red and brawny swelling resembling erysipelas, for which it not infrequently has been mistaken, overspreads the region.

Should a phlegmonous inflammation involve the cellular tissue (*dacryocystitis phlegmonosa*) which surrounds it, the pus burrows in front of the sac, forms pouches in the connective tissue, and in most instances the *lacrimal abscess* thus formed points below the *tendo oculi*. If unmolested, the abscess ruptures externally with the formation of a fistulous opening into the sac, the mouth of which is surrounded by pouting granulations (Fig. 276). Stricture of the lacrimal duct is associated with dacryocystitis.

2. **Prelacrimal Sac Abscess.**—This consists of a swelling above the internal palpebral ligament and a little external to the region of the lacrimal sac, associated with a fistulous opening, from which pus flows, having no connection with the sac itself. It may be caused by a blow at the inner angle of the eye and may be associated with caries and perforation of the lacrimal bone. The same condition appears without injury in children who are the subjects of hereditary syphilis.

The condition is to be distinguished from a true lacrimal abscess by the fact that there is no interference with the passage of tears from the conjunctiva into the sac, and by the absence of acute inflammation. *Prelacrimal sac cysts* are described, and small tumors may appear in this region. One removed and examined by the author had all the histologic appearances of tubercle.

The *treatment* is that of an abscess, together with such constitutional measures as may be indicated by the dyscrasia of which the patient is the subject; cysts or tumors should be removed in the ordinary manner.

3. **Fistula of the Lacrimal Sac.**—This occasionally has been observed as a congenital anomaly, and may be present on only one side or on both sides. The opening is usually directly under the internal palpebral ligament.

Generally a fistulous opening into the sac is caused by the *rupture of a lacrimal abscess*, but it may result from a carious condition of the upper canine teeth. The opening may appear about 1 cm. below the punctum, but also in various spots along a line which runs outward, parallel to the lower orbital border.

It usually communicates with the sac, but in rare instances the opening may lead into the lower canal only, the sac above being shrunken. Pus and mucopus, and later tears, which should descend into the duct, exude from the opening, which for a long time persists as a fine orifice, at the mouth of which appears a drop of clear fluid. This is the so-called *capillary fistula*.

The condition is to be differentiated from a *buccal fistula* below the margin of the orbit, by observing that in the latter the situation is never accurately at the orbital margin, that a sound never passes upward, but only downward, laterally, or posteriorly, and that the secretion is always purulent. Von Szily with the aid of *x*-ray examination has discovered that there may be a communication of the tear sac with the nose, that is, an *internal fistula of the lacrimal sac:* in other words, a spontaneous, false passage. Such a condition may be associated with

empyema of the ethmoid cells. Roentgen-ray studies of the lacrimonasal region are important, as has recently been well shown by Don Campbell, T. M. Carter, and H. Doub. Irregularities, malformations, and the relation of the middle turbinate bone to the lacrimonasal duct may thus be investigated.

4. **Obstruction of the Nasal Duct.**—This always antedates the affection of the sac. It may be situated at any part, but selects by preference the point at which the nasal duct enters into the sac, or the lower end where it passes into the nasal chamber.

In the early stages of catarrhal dacryocystitis there probably is no true stricture of the duct, but the flow from the sac into the nose is prevented by swelling of the mucous tissue; later, and in other instances, cicatricial strictures occur.

Causes of Disease of the Lacrimal Sac and Nasal Duct.—Disease of the lacrimal sac is rarely primary. In young infants so-called dacryocystitis is not infrequently seen—*lacrimal blennorrhea* or *atresia of the newborn*. Donald Gunn thinks that the cause of mucocele of newborn children, becoming afterward dacryocystitis, depends upon a dilated duct, the dilation being brought about during fetal life by obstruction at the lower end, depending, for example, upon some developmental fault. The pus usually contains pneumococci; staphylococci, streptococci, and Bacterium coli are also occasionally present. Many cases depend upon retention of separated cells because of imperforation of the septum between the lacrimonasal duct and the nasal chamber (Zentmayer). In these infants pressure over the sac causes the contained secretion to escape into the conjunctival sac through the punctum; sometimes both sacs are involved; primarily the evidences of acute inflammation are lacking.

Both a local and a general disposition to tear-duct troubles has been assumed by some authors, and by others, for example, Haab, hereditary predisposition has been given etiologic prominence. The female sex suffers more frequently than the male, and the left tear-duct is more often diseased than the right (Cahn).

In the majority of cases blennorrhea of the sac is caused by a retention of the secretion on account of stricture or obstruction in the nasal duct, and the participation of the lining of the sac in an inflammation of the nasopharynx. In other instances strictures result from, rather than cause, the blennorrhea. A proper appreciation of the pathologic conditions of the nasal mucous membrane in relation to diseases of the lacrimal apparatus is of the utmost importance, and in nearly every case of disease of the lacrimal sac and of the lacrimonasal duct morbid conditions of the nasal chambers and of the nasopharynx are present, especially tumefaction of the mucous membrane, hypertrophy, and abnormal position of the turbinate bones, strictures after nasal ulcers, and caries of the nasal bones. Of great importance in this respect, especially in suppurative dacryocystitis, is infection of the ethmoid cells and of the antrum of Highmore, and in a search for a primary cause these regions deserve accurate investigation. In not a few instances

suppuration in the lacrimal portion of the ethmoidal cells has been mistaken for dacryocystitis.

Although it might seem natural that conjunctivitis, and especially purulent conjunctivitis, should cause lacrimal disease, this is by no means frequently the case. Conjunctivitis and blepharitis, so often associated disorders, follow rather than cause the lacrimal affection.

Obstruction of the duct and disease of the sac are sequels of measles, scarlet fever, and especially smallpox, because these exanthems are accompanied by inflammation of the nasal mucous membrane.

Periostitis and caries of the lacrimal bone, the result of syphilis, are important causes. Gummatous growths may block the sac and go on to rapid suppuration. Igersheimer calls especial attention to the frequency with which hereditary lues is a cause of disease of the lacrimal passages in children. *Tuberculosis of the lacrimal sac* is of not infrequent occurrence. It may occur in the subjects of pulmonary tuberculosis or be secondary to tuberculous infection in the neighboring structures. Should there be an admixture of pyogenic bacteria the manifestation may be that of ordinary dacryocystitis (Rollet and Bussy). Trachoma and dacryocystitis are frequently in association and *trachoma of the lacrimal sac* is well known; in such circumstances it becomes friable and difficult to excise (Butler).

The relation between asymmetry of the face and disease of the lacrimonasal duct deserves mention; indeed, Hasner assumed that a local disposition to these disorders depended upon this asymmetry. *Traumatism* accounts for certain cases. Impermeable obstructions follow injuries and the rough use of bougies. Stoppage of the lacrimonasal duct may be caused by pressure from neighboring tumors —for example, in the antrum of Highmore, and by foreign bodies lodged in the lower lacrimal canal and in the nasal chambers. *Actinomycosis* of this region has been reported (von Schroeder).

Fistulas, especially those seen in infants, often arise from disease of the bone, which, in turn, is the result of inherited syphilis.

Tumors of the lacrimal sac are uncommon; epithelioma (Pasetti, Posey), sarcoma (T. H. Butler), and plasmoma (Verhoeff and Derby) have been observed. All excised lacrimal sacs should be submitted to careful microscopic examination.

Prognosis in Lacrimal Disease.—The well-known fact that affections of the tear-passages often stubbornly resist treatment renders a guarded prognosis necessary, which depends entirely upon the condition of the nasal chambers, the duration of the malady, the permeability of the stricture, and the cause of the trouble. If stricture is the result of injury the prognosis becomes especially grave, and the condition may be irremediable. Recently scientific methods of treatment have greatly improved prognosis, particularly because the effects of useless and sometimes reckless introduction of probes are not so frequently in evidence.

Character of the Lacrimal Secretion Under Pathologic Conditions.—The lacrimal sac is a reservoir for the fluid secreted by the conjunctiva, and this fluid is more or less loaded with micro-organisms.

Streptococcus pyogenes, pneumococcus, and other pathogenic organisms are always present in dacryocystitis. They may turn a simple abrasion into a sloughing ulcer or an aggravated hypopyon-keratitis (page 272). They may prevent the healing of an ordinary keratitis, and finally they may inoculate an operative wound and defeat the object of the operation. For this reason it is most important that in any of the three conditions just quoted the permeability of the nasal duct should be ascertained. If it is strictured, it should be opened, and the walls of the lacrimal sac, if inflamed, brought to a healthy condition as speedily as possible, or the sac should be extirpated. The importance of this relation of the lacrimal apparatus to diseases of the cornea and to the prognosis of cataract operations is elsewhere described.

Treatment of Diseases of the Lacrimal Sac and Duct.—Conservative measures are indicated in most of the cases of simple epiphora—viz., intranasal treatment, massage over the sac while the inner canthus is kept filled with an antiseptic liquid, and dilatation of the punctum and irrigation of the sac. Many of these cases are due to ametropia and heterophoria and even to various nervous diseases—for example, tabes dorsalis and neurasthenia. Operative interference is to be deprecated unless the exact cause of the condition is ascertained. Epiphora may be an early sign of exophthalmic goiter (Berger, J. T. Carpenter).

The following procedures are commonly employed: slitting the canaliculus, introducing a probe into the nasal duct, and syringing the sac and nasolacrimal duct; or, in the presence of proper indications, excision of the lacrimal sac or intranasal drainage (page 794). The method of slitting the canaliculus and the introduction of a probe are described on page 792.

After the canaliculus has been dilated or incised, the duct and the sac should be washed out thoroughly with some antiseptic fluid—a saturated solution of boric acid or a 1 : 8000 solution of bichlorid of mercury, or formaldehyd 1 : 3000 or physiologic salt solution. Great care should be taken in using solutions of argyrol and protargol, lest they escape into the surrounding structures and produce unsightly staining of the skin. They should not be injected into the sac. Mercurophen (1 : 8000) and mercurochrome (1 per cent.) may be used in place of the previously named solutions; they must not be allowed to enter adjacent tissues.

Some surgeons, as a rule, split the upper canaliculus, although the usual practice is to approach by means of the lower passage. If there is much distention of the sac, it has been suggested to enter the upper passage and incise both this and the wall of the sac.

In making use of probes, it is advisable to begin the first trial with a No. 1 conical probe (Bowman's or Williams'); if this fails, a smaller one may be tried. Either rapid or gradual dilatation is employed, the latter being the preferable method. Undue efforts should never be used, as it is extremely easy to make a false passage and perforate the delicate structure of the lacrimal bone, while roughness in the use of probes, by scraping off the mucous membrane, may cause the most impermeable type of stricture.

Often it is not necessary to use probes at all. The point of an Anel syringe charged with a physiologic salt solution or an antiseptic lotion may be readily introduced through the punctum into the canaliculus until it reaches the entrance into the sac which is thus thoroughly washed out. Should the fluid pass out through the nose it is evident no material obstruction exists in the lacrimonasal duct. This maneuver is materially assisted if a drop of adrenalin solution (1 : 1000) is instilled with the cocain prior to the operation.

Sounds should be used at first every second or third day, but as the case progresses longer intervals may elapse. Large probes (4 mm. in diameter) are advocated by Theobald, but, in the author's experience, are not essential. Ziegler, employing a dilator which he has designed, rapidly dilates the duct not only in cases of obstruction, but when it is desired to increase drainage from the conjunctival sac.

If a lacrimal abscess supervenes and is seen early, the canaliculus should at once be slit and, if possible, the secretion evacuated and the passage into the nose restored. Frequently the pain and swelling are such as to render this impossible, and the opening must be made upon the face, about 1 cm. below the palpebral tendon, cutting downward and outward. The cavity should then be thoroughly cureted, packed with gauze, and allowed to heal gradually from the bottom.

An excellent practice is to use hot compresses over the swelling, preferably of carbolized water, at a temperature of 120° F., frequently changed and applied for five or ten minutes at a time. Later the passage into the nose may be rendered patulous, in the manner already described. The practice of introducing a lead or silver style the author has abandoned, although many surgeons are strongly in favor of its use, especially the use of a lead style, which is preferable to a cannula (H. Moulton).

The treatment of dacryocystitis of infants should consist in the use of a simple collyrium, boric acid or boric acid and sulphate of zinc, and frequent evacuation of the contents of the sac by pressure, and gentle massage. In the experience of the author this is usually sufficient; occasionally slitting the canaliculus and passing probes may be necessary. If pneumococci are present in the secretion, mercurophen (1 : 8000) is valuable; mercurochrome may also be used. Argyrol is also of service especially because of the ease with which it passes through the canaliculus into the sac, especially if a few drops are placed at the inner commissure and the surface over the sac is gently massaged.

Swelling over and around the lacrimal sac, with fistulous communication into it, occasionally disappears under the influence of a pressure bandage renewed at proper intervals.

In addition to the local measures already mentioned, solutions of nitrate of silver and salicylic acid, and of iodoform, aristol, and creolin (1 per cent.) have been advocated.

In acute inflammation with abscess formation, quinin and iron in the form of Basham's mixture are indicated; in syphilis, with disease of the bone and gummatous deposit, the usual drugs should be ex-

hibited; indeed, it is important to make a Wassermann test in stubborn cases of chronic dacryocystitis, even though no nasal deformity (saddle-nose) or ozena be present. In so-called struma, cod-liver oil, hypophosphites, and preparations of iron are the most trustworthy remedies.

Scrupulous attention to the nose and the nasopharynx is necessary. Good results follow simple spraying of the parts with Dobell's solution or peroxid of hydrogen one-third, water two-thirds, while carrying on the regulation measures for the relief of the lacrimal disorder. If there is decided disease of the region, the proper treatment often requires the usual procedures of nasal surgery. In children adenoids should be removed. The importance of examining accessory nasal sinuses has been pointed out, especially the ethmoid and the maxillary sinus. The value of *x*-ray examination has been referred to.

Occasionally, although a duct has been thoroughly opened, the probe passes readily, and the liquid used in the syringe flows freely from the nose, the epiphora continues, and the eye fairly swims in tears. In such circumstances a probe should be passed into the nose and the entrance of the duct into the inferior meatus properly exposed by means of a nasal speculum. Quite often it will be seen that a thickening of the duct entrance, or perhaps a valve-like flap of mucous membrane, occludes the passage. This is pushed aside by the probe or forced aside by the liquid when it is injected, but entirely prevents flow of tears into the nose.

If a fistula remains, this may sometimes be closed, as already stated, by compression. In the event of failure, freshening of the edges and the galvanocautery may be tried, the surrounding pouting granulations being removed by scraping. The capillary fistulas are productive of no inconvenience and may be allowed to remain undisturbed.

Extirpation of the lacrimal sac is indicated, and usually yields good results in cases of chronic dacryocystitis. It may be employed if conservative and ordinary surgical measures have failed, if the patient cannot or will not devote sufficient time to treatment, if there is an impassable stricture, if an operation on the eyeball is speedily necessary, if there is a serpiginous ulcer of the cornea, and in cases of caries of the lacrimal bone. The operation is further indicated in those whose occupation exposes them to corneal injury (Axenfeld) and in insane patients. The operation is so satisfactory in its results that, in the author's opinion, it should in large measure replace the use of probes and the other measures which have been described. If, subsequently, the epiphora is annoying, *extirpation of the lacrimal gland* has been performed, and was especially advocated by C. R. Holmes. In place of complete excision, removal of the palpebral gland may be tried. The author has not found it necessary to employ either of these procedures for this purpose. (For methods of operating see page 794.) Usually, however, as the conjunctiva resumes its normal condition, the epiphora, under ordinary conditions at least, ceases to be annoying, and often disappears. In place of excision of the lacrimal sac other methods of operating (intranasal drainage) in cases of dacryocystitis are preferred by many surgeons (page 796).

CHAPTER XX

DISEASES OF THE EYEBALL AND ORBIT

Congenital Anomalies.—*Anophthalmos,* or complete absence of one or both eyes, is an affection which, like the other congenital anomalies, more frequently is double than one sided. A child born without eyes may be healthy and well developed in other respects, or may be the subject of additional congenital deformities. The palpebral fissures are small, the lids usually deficient in size, sunken, and upon their separation the empty orbit is revealed. Usually (always, according to some authors) careful dissection will expose a rudimentary eyeball at the apex of the orbit. Sometimes *cysts* of bluish hue are connected with rudimentary eyes, the cyst being evident in the lower part of the orbit or in the lower lid—*orbitopalpebral cyst.* Retinal elements are present in a cyst of this character.

The most reasonable explanation of this anomaly is that no primary optic vesicle has budded out from the anterior primary encephalic vesicle, or that, having budded out, it has failed to form a secondary optic vesicle.

Microphthalmos and *megalophthalmos* are anomalies of the globe to which reference has been made.

Cyclopia is a congenital malformation characterized by a fusion of the orbits and the two eyes in the middle of the face, so that there is only one eye situated in the place normally occupied by the root of the nose.

General Symptoms of Orbital Disease.—Two symptoms are so constantly present that they may be said to be essential in the clinical picture of most of the affections of the orbit:

1. *Proptosis or Exophthalmos.*—This consists of more or less protrusion and displacement of the globe.

2. *Immobility of the Eyeball.*—This may be complete or partial, and, if vision is unaffected, the limitation of the movements of the eye is associated with diplopia.

The following signs may also be associated with orbital disease:

(*a*) *Chemosis of the conjunctiva,* either universal or else localized upon a special portion of the globe, indicating the neighborhood of the diseased area.

(*b*) *Redness, swelling,* and *edema* of the eyelids, especially in inflammatory affections of the cellular tissue of the orbit and disease of the accessory nasal sinuses.

(*c*) *Pain,* most noticeable when the patient attempts to move the eye or when the surgeon palpates the globe and presses it inward. In addition to the pain in the orbit itself, *frontal headache* is a common symptom, especially if the sinuses are involved, and *tenderness on pres-*

sure along the margin of the orbit and accessible portions of its walls is one sign of disease of the periosteum.

(*d*) *Fluctuation* occurs, but not constantly, if an abscess of the orbit has formed.

(*e*) *Disturbance of Vision.*—In some cases of orbital diseases there is no disturbance of vision; in others there may be marked changes in the eye-ground—papillitis (papilledema), atrophy, hemorrhages, and vasculitis or perivasculitis.

Periostitis of the orbit is both *acute* and *chronic*, and in the acute type appears either as a *localized* affection or as a *diffuse* suppurative process.

The **symptoms** of acute localized periostitis are pain, tenderness over the seat of the disease, usually the margin of the orbit, injection and chemosis of the conjunctiva, and some swelling of the lids and protrusion of the ball. In the diffuse variety of the disease all the foregoing symptoms are much aggravated, and there may be, in addition, fever, general headache, delirium, and stupor. Periostitis of the roof of the orbit is fraught with special danger on account of the proximity of the cranial cavity. In such a case the differential diagnosis between it and an orbital cellulitis becomes extremely difficult. In fact, the cellular tissue is associated with the periosteum in the inflammation. A *subperiosteal abscess* may form and become encapsulated or, passing forward, burst through the skin of the eyelid or at the angles of the orbit and form an *orbital fistula*. Other situations of an abscess may be between the periosteum and the muscles, within the muscle cone and in the orbital fat (see also page 661).

In chronic periostitis there are deep-seated pain, often worse at night, tenderness on pressing the eyeball backward, thickening of the tissue beneath the orbital margin, and swelling of the lids and conjunctiva, although the latter symptoms, together with proptosis, may be absent.

According to Mraček, syphilitic periostitis most frequently attacks the orbital margins, and may occur in a *gummatous* or a *sclerosing* form. It less commonly involves the orbital walls behind Tenon's capsule, and is then generally gummatous in type. The site is usually in the upper or outer wall, and the disease causes trigeminal neuralgia, worse at night, and restriction in the mobility of the globe, with squint and diplopia. Optic neuritis may occur. Tuberculous periostitis, especially in children, is usually situated at the upper and outer or lower and outer orbital margins.

The **causes** of periostitis, especially of the chronic form, in addition to syphilis, in which disease it is sometimes a secondary, but more often a late, manifestation, are rheumatism, tuberculosis, injuries and affections of the sinuses, notably the frontal and the ethmoid. Syphilitic periostitis is more common in adults than in children, but may attack the latter (*periostitis with hereditary syphilis*).

The **prognosis** depends upon the type of the disease. If localized, this is favorable; if diffuse and suppurative, not only may extensive

implication of the tissues surrounding the globe leave permanent disabilities and deformities (exophthalmos, muscle palsy, optic-nerve atrophy, necrosis), but the inflammation may extend to the meninges of the brain and cause death.

Chronic periostitis may last for months, and in any type fistulas, necrosis, and caries of the bone are the common result. Periostitis due to syphilis presents the most favorable prognosis.

Treatment.—The constitutional treatment depends upon the cause, and includes the iodids and salicylates in rheumatic cases, and the use of mercurials and salvarsan in syphilitic cases.

The surgical treatment of acute periostitis consists in an incision into the affected area and evacuation of the pus; in short, the treatment is the same as that applied to acute periosteal disease elsewhere located. The relation of periostitis to sinus disease demands a careful examination of the sinuses and treatment according to the findings.



FIG. 277. –Caries of the orbit and ectropion (from a patient in the University Hospital).

Caries and Necrosis.—Caries is prone to attack the margin of the orbit, especially the lower and outer part, and may be due to syphilis or tuberculosis. An injury often is the exciting cause.

The **symptoms** of periostitis are present, suppuration develops, the abscess comes to the surface through the lid over the diseased area, rupture occurs, with the discharge of pus, a fistula forms, surrounded by granulations, and through this a probe will detect the softened bone. Very decided deformity of the lid may be occasioned, most commonly in the form of an ectropion (compare Figs. 98 and 101).

Caries of the orbit is most common in children, and, as has been pointed out, selects the margin of the orbit for its site, although it may occur in the roof, in which case it becomes a complication endangering life, owing to the proximity of the brain. The inflammation may spread to the orbital tissues and cause exophthalmos and neuroretinitis.

Necrosis of the orbit is much less common, and its immediate cause is an osteitis occurring as a consequence of acute periostitis. A fragment of bone completely separated by a fracture from the periosteal surroundings would probably undergo necrosis, and the rough use of probes may cause mortification of the delicate lacrimal bone. Necrosis, unlike caries, is more common in adults.

Treatment.—This consists of the remedies recommended in the treatment of periostitis, and, as caries is a chronic affection and most common in strumous (tuberculous) subjects, cod-liver oil, phosphates, and iodid of iron should be included in the constitutional measures, and should be exhibited for long periods of time.

The local treatment during the early ulcerative stage of caries consists of evacuation of foci of suppuration, careful cleansing with antiseptic solutions, and drainage. Considerable caution is necessary before resorting to the removal of the diseased bone with a gouge, because the process is essentially chronic and may be aggravated by the manipulations of the instrument; but roughened bone should be scraped with a sharp spoon and the diseased portions thoroughly removed. If the roof of the orbit is affected, great care is necessary lest the cranial cavity be penetrated. If a piece of the orbital wall has undergone necrosis, this should be removed when it has become loose or detached. The regions overlying the sinuses should be carefully examined.

Cellulitis (*Phlegmon of the Orbit*).—There are several varieties of inflammation of the cellulofatty tissue of the orbit. Thus the inflammation may be acute, subacute, or chronic, unilateral or bilateral, and finally it may undergo resolution or, as more commonly is the case, terminate in suppuration.

In the *mild* form the *symptoms* are dull pain, swelling of the lids, slight exophthalmos and diplopia, without inflammatory symptoms and without constitutional disturbance.



FIG. 278.—From a photograph of a patient in the Philadelphia General Hospital suffering from double orbital cellulitis the result of erysipelas.

In the *acute* phlegmonous variety of the disease there are chills, fever, deep-seated pain, most marked upon attempting to move the eyes, general headache, exophthalmos, limitation in the movements of the eye (which may become entirely fixed), and swelling and edema of the lids, together with hyperemia and chemosis of the conjunctiva. The last two symptoms are so severe at times as to give at first sight the general impression of a violent attack of purulent conjunctivitis (Fig. 278).

In the earlier stages vision is not usually affected, but later there may be optic neuritis followed by atrophy, dilatation of the pupil, anesthesia, and even ulceration of the cornea, and, indeed, in severe cases the eyeball may suppurate. In certain types of orbital cellulitis extensive intra-ocular changes occur, with hemorrhages and vascular alterations, due to compression of the central vessels of the retina

producing stoppage of the circulation and edema and exudation into the retina (H. Knapp). Blindness from orbital abscess may be due to retrobulbar necrosis of the optic nerve, caused by thrombosis of the pial vessels and of the central vessels (Bartels). Fluctuation finally develops; pointing usually occurs below the inner portion of the supra-orbital ridge.

The symptoms of *chronic* abscess are much less violent and distinctive than those just described. They may be mistaken for other morbid conditions, especially as the abscess is commonly associated with diseased bone or periosteum in scrofulous subjects, or may occur in them from an injury or the presence of a foreign body.

The **causes** of orbital cellulitis are various. It may be traumatic in origin, infection being conveyed through a wound, especially if associated with the entrance of a foreign body; it may follow in the wake of scarlatina, measles, typhoid fever, or influenza; or it may be the result of meningitis. The most violent types of orbital cellulitis occur with facial erysipelas. In these instances the affection is usually double. The extension of inflammation from diseased teeth or suppuration in the ethmoid cells, sphenoid, or antrum of Highmore may cause the affection. Birch-Hirschfeld's investigation demonstrates that the largest number of orbital inflammations (about 60 per cent.) are due to accessory sinus inflammation. The infection is conveyed from the sinus to the orbital contents by means of septic thrombosis, thrombophlebitis, lymphangitis, or erosion of the bony partition. Finally, a certain number of cases are *metastatic*, and develop in the course of pyemia, especially puerperal septicemia. During the past war a number of cases of metastatic orbital abscess occurred in association with osteomyelitis, chiefly of the femur. Some of them began with fixation of the globe, slight exophthalmos, but with no inflammatory symptoms evident for several days. The association of orbital cellulitis with periostitis has already been referred to, and a certain amount of cellulitis occurs whenever there is a general inflammation of the globe.

Progress and Prognosis.—In mild cases the prognosis is favorable; in severe cases, unfavorable; and in double cases, especially those which have originated under the influence of erysipelas, usually fatal. Although the pus may make its exit through the conjunctiva or eyelid, it may also pass backward through the sphenoid fissure. In pyemic cases, and, indeed, in the course of any severe inflammation of the cellulofatty tissue of the orbit, *phlebitis of the orbital veins* may become a complication and extend to the cavernous sinus, leading to a fatal termination. If the disease passes to the cavernous sinus upon the opposite side, the other eye also becomes involved and exophthalmos is evident.

In framing a prognosis it is necessary to consider the effect of the disease upon the eyesight and upon the life of the patient. Sight may be impaired or destroyed by the development of optic neuritis, optic-nerve atrophy, exudation and hemorrhages into the retina, or by sup-

puration of the cornea; life may be endangered by an extension of the suppurative process into the cranial cavity, or by the original malady which caused the cellulitis.

Treatment.—The general treatment should include supporting measures and iron and quinin. Occasionally the pus points in the conjunctival sac and may be evacuated by an incision through the conjunctiva between the ball and the side of the orbit, care being taken not to injure the ocular muscles and to secure good drainage afterward. Sometimes, according to the indications, the abscess may be reached, for example, by an incision through the skin surface placed just within the lower orbital margin. In deeper situations, that is, if the trouble starts from the bone or paranasal sinuses, the purulent focus is best reached by a curved incision made over the orbital ridge which divides the periosteum, which is next separated with an ordinary bone elevator, kept well between the bone and the periosteum, thus avoiding the levator, the tendon of the superior oblique, and the lacrimal gland. If pus does not immediately present, the depth of the orbit must be explored with a probe until the pocket of pus is found, and evacuated by an incision through the periosteum. Drainage may be secured by means of iodoform gauze or an ordinary drainage-tube. The position of the original incision is determined by the probable situation of the pus; that is to say, whether it shall be made along the upper, lower, inner, or outer orbital margin. If the source of the pus is from the ethmoid, the orbital plate of this bone should be perforated, carious bone and necrotic tissue removed, and a drainage-tube carried from the orbit, through the ethmoid, into the nose, which not only secures an adequate drainage, but permits the subsequent washing out of the tract. Indeed, the frequent association of sinus disease with orbital cellulitis usually demands that the incision shall be so placed and sufficiently broad to render exploration of the orbital walls practicable and treatment of the affected sinus (frontal or ethmoid) possible.

Inflammation of the Oculo-orbital Fascia (*Tenonitis*).—This affection is characterized by swelling of the upper lid, the discoloration of the tumefied lid being limited to its tarsal portion (Pincus); pain on the slightest movement of the eye, some proptosis and limitation of movement, together with the appearance of a watery nodule or vesicle situated over one of the recti muscles; in other cases the chemosis may be more general. Primary serous tenonitis is a rare disease, about 40 cases being on record (Birch-Hirschfeld). Tenonitis may follow an injury or an operation—for instance, tenotomy; in some instances it is due to rheumatism, focal infection (teeth, tonsils, intestines), possibly to tuberculosis, and it has been noted as a sequel of diphtheria, typhoid fever, and epidemic influenza, and may be caused by syphilis. Tenonitis occasionally complicates severe iridocyclitis, and practically always accompanies panophthalmitis. Lesions almost similar to those of tenonitis are occasionally caused by an intra-ocular growth; for example, a sarcoma (see page 404).

The treatment should consist of warm fomentations and, according

to the indications, iodid of potassium or the salicylates. Areas of focal infection should be eliminated.

Thrombosis of the Cavernous Sinus.—Cavernous sinus thrombosis may be divided into: (1) those cases which depend upon debilitating conditions, and which may occur in very young life, or in old age, the so-called marasmic types, and which nearly always follow an involvement of the longitudinal or lateral sinus; (2) those which are due to injury, for example, fracture of the skull, or operations in the neighborhood of the gasserian ganglion, the traumatic types; (3) those which are due to an infection, usually secondary to a thrombophlebitis of an afferent or efferent vein, but which also primarily may be due to an extension from meningitis, or to a suppurating area in the sphenoid or temporal bone.

The thrombotic process may be septic, the most common form, or aseptic, a purulent condition being absent. One or both sinuses may be involved, the bilateral process being a frequent one. Because of the elaborate venous anatomic connections of the cavernous sinus there are many areas which, if they contain foci of infection, may send their septic products to the sinus, which determine the thrombosis. Thus, the infection may proceed from scalp wounds, from the neck as the result of a carbuncle, from the eyelids because of furuncles, styes, and pustules, from pimples and infected wounds in the facial area, from erysipelas, from ulceration of the mucous membrane of the nose, and from infected hair follicles in its aperture, from the paranasal sinuses, especially the sphenoid and ethmoid, from tooth-root abscess, carious teeth, and septic tonsils, from suppurative otitis media and mastoiditis, and from the orbit on account of cellulitis, abscess, and periostitis.

Next to disease of the sphenoidal sinus, pyogenic infection from the ear is the most common cause of thrombosis of the cavernous sinus (St. Clair Thomson), but a number of cases have been traced to foci of infection in the facial area, almost as many, in fact, as have arisen from aural disorders, and sometimes the source of the infection is an extremely trifling one, a small pimple on the face, or an infected hair follicle in the nose. In acute septic thrombosis there are chills, septic temperature, nausea, vomiting, mental disturbances, and coma. Should lumbar puncture reveal a cloudy fluid, meningitis is a complicating factor, but the important symptoms are ocular in their manifestation, and are due to venous stasis and involvement of the nerves which pass through the sinus.

Early prodromal symptoms may be headache, with or without fever, pain in and around the eyes and in the teeth, and diplopia. Increasing exophthalmos, with edema of the lids, face and root of the nose and of the conjunctiva supervene, first on one side, and soon (in over half of the cases) on the opposite side, in most instances within thirty to forty-eight hours. Ptosis of paralytic origin is common, although it may be difficult to decide whether the drooping of the lid is due to paresis of the third nerve or to edema. According to the muscle involved there may be divergent or convergent strabismus, and finally, complete

fixity of the eyeballs, when all the nerves, third, fourth, and sixth, are implicated.

Conspicuous ophthalmoscopic signs are edema of the retina and great distention and tortuosity of the retinal veins. A true optic neuritis indicates orbital involvement (thrombophlebitis of the orbital veins). More often the evidences are those of papilledema, that is, moderate choked disk. Retinal hemorrhages are not uncommon, but ophthalmoscopic signs, although probably evident sooner or later, have not always been discovered. Owing to implication of the fifth nerve, there is anesthesia of the cornea, and this tissue very early in the disease may become cloudy, and ulceration may develop. While ocular manifestations of sinus thrombosis may be mistaken for exophthalmos from other causes, for example, injury, fractures of the skull, and orbital tumor, the history and symptoms are so distinctive that such an error should always be avoided. Orbital cellulitis is not so readily differentiated; the absence of cerebral symptoms in the orbital affection (in most of the cases) would be an important distinguishing feature. The *prognosis* of infected cavernous sinus thrombosis unless drainage can be secured, is almost always fatal. The feasibility of drainage has been demonstrated (Hartley, Ballance, and others). The approach has been through the Hartley-Krause gasserian ganglion route, through the orbit (advocated by Mosher), and through the petrous portion of the temporal bone (Borden).

Tumors and Cysts of the Orbit.—Tumors have been divided by systematic writers into those which originate in the orbit, but are unconnected with the globe of the eye; those which arise from the periosteum or bony walls of the orbit; those which commence in the cavities close to the orbit; and those which originate in some vascular disease within the cavity of the orbit or the neighboring portions of the cranial cavity, and which usually give rise to *pulsating exophthalmos.*

Two classes of tumors, namely, those which arise from the optic nerve and those which arise from the lacrimal gland, are sometimes included among the orbital growths. They have been discussed in another section.

The *nature* of orbital tumors is either benign or malignant, and they may be congenital or acquired, primary or metastatic. After enucleation of an eyeball for sarcoma of the choroid there may be a recurrence of the growth in the orbital tissue.

Symptoms.—These vary according to the position, size, and density of the tumor, but in general terms are those which have been narrated as more or less common to all diseases of the orbit. Small tumors situated in the posterior part of the orbit may be slow of growth and exist for a long time, even years, without marked symptoms of their existence. Ultimately proptosis and swelling of the lid develop; occasionally a tumor can be felt by deep palpation. With respect to the protrusion it may be said that a tumor within the cone of the recti muscles is apt to cause a forward displacement of the globe, while one situated outside of this cone may displace the eyeball in some particular direction.

Considerable proptosis may occur under the influence of an orbital tumor without causing the globe to protrude between the fissure of the lids. This is due to the fact that the lids are extensible and accommodate themselves to the increasing volume behind them; finally, however, the protrusion may be so great that the lids can no longer close over the prominent ball.

Prognosis.—This depends upon the nature of the tumor, the density of its tissue, the rate of its growth, and the availability of surgical interference.

Treatment.—Benign tumors, cysts, and some encapsulated sarcomas can be removed, according to the methods described elsewhere (page 753) without sacrificing the eyeball, but the treatment of the majority of malignant growths requires exenteration of the orbital contents; afterward radiation (*x*-rays or radium) should be employed. These measures are also useful in the event of a recurrence of the growth, and may be used with success in the treatment of orbital growths independently of surgical interference (page 667). As was pointed out by Panas, Snell, and others, certain tumors of the orbit, probably lymphomas, occasionally disappear under medicinal treatment—for example, iodid of potassium, arsenic, etc. Hence the necessity of careful medication before surgical measures are tried. The difficulty of distinguishing an orbital tumor from a chronic inflammatory process is often great and no method of diagnosis should be omitted, especially careful examination of the accessory sinuses. In this respect *x*-ray examination is important.

1. **Tumors which Originate in the Tissues of the Orbit.**—These include cysts, fibromas, neurofibromas (Stieren, W. Parker), cavernous and simple angiomas, lymphangiomas, lipomas, enchondromas, lymphomas, cylindromas, endotheliomas, psammosarcomas, and the various other types of sarcoma. Discrete lymphoid infiltration of the orbit, causing proptosis, has been reported by Coats. Carcinoma, except in connection with the lacrimal gland, does not occur in this situation as a primary tumor; it may, arising from the lids or conjunctiva, grow inward and involve the orbit (page 189). In a total of 68 cases of metastatic carcinoma of the choroid there was in 13 of them an extraocular extension of the growth from the focus in the choroid (Shumway). Metastasis of carcinoma from distant organs to the ocular muscles has been described (see page 617). Adrenal tumors in children, usually under four years of age, with metastasis to the orbit, have been reported (Quakenboss, Verhoeff). Metastasis from the thyroid (metastatic thyroid tumor) has been observed (A. Knapp).

Sarcomas of the orbit may be primary or metastatic and may present the various types of cellular structure characteristic of these tumors. Some sarcomas of the orbit should be classified with the *endotheliomas*. If the morbid process is an extensive one, radical removal of the entire contents of the orbit is the only procedure, and subsequently *x*-rays or, preferably, radium should be employed. Even in inoperable malignant disease of the orbit pain is lessened by

the application of the *x*-rays, and the complete disappearance of sarcoma of the orbit, without operation, under the influence of repeated applications of Roentgen rays has been reported, and represents a therapeutic measure deserving of the most thorough trial. In like circumstances radium may be used and is an even more serviceable agent. Encapsulated sarcomas may occasionally be removed with preservation of the eyeball. *Traumatic sarcomas* offer a most unfavorable prognosis, and operation hastens rather than retards the fatal issue. Sarcomas of the orbit should not be confounded with those which arise within the eyeball and have burst their boundaries (see page 404). Although tuberculosis of the orbit usually manifests itself at its margin as a caries and as a tuberculous periostitis, a rare condition discussed by Birch-Hirschfeld is a *tuberculosis of the retrobulbar tissue* which has been mistaken for a sarcoma. The primary focus of such tuberculosis may be in the sphenoid and in the meninges, and



FIG. 279.—Metastatic sarcoma of the orbit (from a patient under the care of Dr. Wharton in the Children's Hospital).

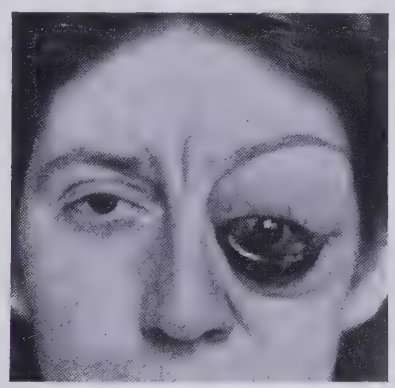


FIG. 280.—Sarcoma of the orbit springing from the periosteum over the great wing of the sphenoid.

he insists that in all orbital tumors the possibility of a tuberculous process must be considered.

Berlin divided *orbital cysts* into two principal groups, *cephaloceles* and *true cysts*. Cephaloceles are located at the root of the nose, and extend to the brow, nasal cavities, or orbit. Characteristic of cephaloceles and meningoceles is the fact that they present at the inner side of the orbit, that they fluctuate and are transparent. True cysts may be classified as cysts from constriction, which are derived from congenital meningoceles (Klingelhoffer); as extravasation-cysts—that is to say, blood-cysts, hematomas, etc.; as exudation-cysts, which are very rare; as dermoids, which are the most frequent cystic tumors growing in the orbit, and as oil and serous cysts which are derived from them; mucous cysts, which may communicate with the nose, and as echinococcus cysts. Extravasated blood in the retrobulbar tissue may become encapsulated and simulate a blood-cyst, and subperiosteal

blood-cysts have been described (Denig, Lamb); rarely a cholesterin cyst arises from the bones of the orbit (Benedict).

Incision into a simple cyst followed by irrigation with an astringent or antiseptic solution has been practised, but the proper procedure is to expose it and dissect it intact from its bed (see page 753). Care must be taken not to confound a cephalocele with an orbital cyst.

2. **Tumors which Arise from the Periosteum or Bony Walls of the Orbit.**—These include:

(*a*) *Sarcomas*, which arise from the periosteum.

(*b*) *Thickening of the periosteum*, which may simulate a true tumor, especially if the underlying bone is hypertrophied (hyperostoses: these may be multiple or diffuse), and—

(*c*) *Exostoses.*—The latter are very hard tumors having an ivory-like shell and a nucleus of spongy bone, their anatomic structure in general being like that of the osteomas proceeding from adjacent cavities.[1] All orbital osteomas grow slowly—the external exostoses more slowly than the bony tumors which originate from the frontal and ethmoid sinuses. They spring from the periosteum, and are generally found at the upper border of the orbit, although they may occur at any portion of the orbital border, and are recognized by their dense hardness and evident connection with the bone and by *x*-ray examination.

They may arise from injury; sometimes they are congenital, and often their origin is obscure. Occasionally a sarcoma (a psammo-sarcoma, as in a case under the care of the author) may be completely encased in a thin osseous capsule and simulate an exostosis (Fig. 379).

The operation for the removal of an exostosis, after its exposure by suitable incisions of the soft parts and periosteal covering, consists in drilling it away at the base and completing the separation by means of a hammer and chisel. An electric drill and saw is of special service in these operations.

3. **Tumors which Arise in Cavities or Tissues Close to the Orbit.**—These include:

(*a*) *Encephalocele*, a rare condition, which appears in the form of a somewhat pulsating, fluctuating protrusion at the inner angle of the orbit; it is of congenital origin.

(*b*) *Nevi, epithelioma*, and *lupus*, which may extend from the skin of the face into the orbit.

(*c*) *Polypi* from the nasal chambers and surrounding sinuses, and—

(*d*) *Osteomas* of the frontal and ethmoid sinuses.

An *osteoma* consists of a dense growth, with predominance of the ivory shell, and only a trace of spongy tissue (occasionally the reverse occurs). Generally the surface is covered with a delicate connective-tissue envelope, and part of this may be the seat of polypoid growths coming from the remains of the mucous membrane which atrophies under pressure of the tumor.

According to Andrews, osteoma of the frontal sinus first makes

[1] For a valuable paper by J. A. Andrews, on "Osteomas of Orbit," see Medical Record, September 3, 1887.

its appearance by a tumor at the upper inner angle of the orbit, and may be associated with the formation of polypi and suppuration of the sinus. One which grows from the ethmoid sinus first appears at the inner angle of the orbit, and the eyeball is displaced laterally.

If an osteoma springs from the antrum of Highmore, the tumor appears behind the lower eyelid, and the eyeball is displaced upward; if it arises in the sphenoid fissure, sight is affected by compression of the optic nerve.

Extirpation of osteomas in the sinuses is attended with considerable risk, and a number of fatal cases are on record.

4. **Tumors which Originate in Some Vascular Diseases Within the Cavity of the Orbit or in the Neighboring Portions of the Cranial Cavity (Pulsating Exophthalmos).**—Under the name *pulsating exophthalmos* a number of conditions of diverse origin have been described, and more than 300 cases are now on record. The conspicuous symptoms which may arise in the course of this disease, although, naturally, not all of them are present in each case, are as follows: Exophthalmos, most frequently with the eye displaced outward and downward; bruit, usually heard over the eye and above the orbit, but sometimes audible over the whole skull and evident to the patient as a roaring, humming, buzzing, or hissing sound; pulsation, which may be visible or demonstrable only by palpation, or by pressing the globe backward into the orbit; distention of the veins at the inner angle of the orbit, especially enlargement of the angular vein, and of those of the lid and even of the forehead and on the surface of the conjunctiva; corneal complications, usually in the form of exposure keratitis; frequently hyperemia of the iris and rarely actual iritis; commonly hyperemia of the nerve-head, and, occasionally, optic neuritis and even choked disk; frequently marked distention of the retinal veins and scattered retinal hemorrhages; disturbances of ocular motility, sometimes so extensive as to implicate all of the exterior ocular muscles, sometimes only one or other of them, the external rectus being the one most frequently affected where a single muscle is involved; occasional involvement of the trifacial, of the facial, and disturbances of taste, smell, and hearing. The association of glaucoma and pulsating exophthalmos has been observed (Elschnig).



Fig. 281.—Pulsating exophthalmos (from a case under the care of Dr. Kent Wheelock, Fort Wayne, Indiana).

Formerly such symptoms were regarded as evidence of true aneurysm of the ophthalmic artery, but pulsating exophthalmos may also be due to a vascular tumor or an intracranial affection. As Rivington demonstrated, the affection may be caused by an extra-orbital aneurysm of the ophthalmic artery, aneurysm of the internal carotid, or an aneurysmal varix involving the internal carotid and the cavernous sinus. The last-named lesion—arteriovenous communication—is the one most frequently responsible for these phenomena. Dilatation from obstruction of the ophthalmic vein may cause the condition, but aneurysm by anastomosis, which may involve the orbit by spreading from neighboring parts, is not accompanied by exophthalmos. Traumatism is responsible for the majority of the cases, being the essential cause in about 60 per cent.

Treatment.—This has included: (1) Ligation of the larger arteries of the neck; (2) operations upon the orbit; (3) compression of the common carotid; (4) direct compression of the venous swellings of the eyelids and the angle of the orbit; (5) gelatin injections; (6) the administration of certain drugs and rest in the recumbent posture. Of these various procedures, ligation of the common carotid and orbital operation furnish the most satisfactory results. But ligation of the internal carotid alone or of it and the external carotid has been performed. The latter operation is preferred by Reid. According to the investigations of the author and Holloway in 1907, ligature of the common carotid had been performed one hundred and fifty times, with cure or improvement in 64.6 per cent., failure in 25.3 per cent., and death in 10 per cent. of the cases; in a certain number of them, about 10 of the total number, both carotids had been ligated. The orbital operations have included ligation of the superior ophthalmic vein, of the inferior ophthalmic vein, of the angular vein, and of the smaller orbital veins, and the results in most of the cases have been good. If ligature of a common carotid fails before a second carotid is tied, the operation of dissecting out and tying the distended superior ophthalmic vein may be performed. If there is a distinct venous swelling in the orbit, with evident distention of the angular or superior ophthalmic vein, as the operation of choice isolation, ligature, and resection of this venous channel has been recommended. But it is not without danger, and the author is aware of one fatal case, death being due to extension backward of a thrombus into the brain. Compression of the common carotid, with or without the administration of iodid of potassium, has been successful in a few instances, and may be tried before radical surgical means are resorted to.

Exophthalmic Goiter (*Graves' Disease; Basedow's Disease*).—This disease, when it is perfectly developed, is characterized by three cardinal symptoms—enlargement of the thyroid gland, palpitation of the heart, and prominence of the eyeballs. As the affection should be classified with diseases of the ductless glands, the student is referred for a full consideration of the subject to treatises upon the practice of medicine.

Inasmuch, however, as one of the cardinal symptoms—prominence of the eyeballs—is a very marked one, and as there are certain changes seen especially in and around the eyes, a few words may be added. Exophthalmos varies from a mere prominence of the eyeballs, such, for instance, as is noticeable in a highly myopic globe, to a degree of protrusion so great that the eyelids are unable to close. The exophthalmos has been attributed to engorgement of the orbital vessels, caused by excitation of the sympathetic, to increase of the orbital fat and edema of the muscles, and to irritation of the sympathetic, which produces spasm of the unstriped muscle-fibers (*Müller's* or *periorbital muscle*). Excessive epiphora may be present as an early symptom—*i. e.*, before exophthalmos appears (Berger). The lids may be pigmented and eversion of the upper lid is often difficult (Gifford's sign), due perhaps to tonic contraction of the superior tarsal muscle or else to edema of the lid, which is not uncommon. Four important ocular symptoms or signs are the following:

1. *Von Graefe's sign*, which is very important in the early recognition of the disease. Normally, when the globe is turned downward, the upper lid moves in perfect accord with it; in this disease, on rolling the eyeball downward, the upper lid follows tardily, or does not move at all. The symptom is not always present, but it may be noted prior to the development of exophthalmos or at least when it is present only in a trifling degree, and it persists after the protrusion of the eye has subsided.

2. *Stellwag's Sign.*—This consists of imperfect power of winking or diminished frequency in the act; thus, there may be a number of rapid winks, succeeded by a long pause in which there is no movement of the lids, or each time that nictitation occurs it is not complete, and the margins of the lids do not, as in the normal eye, come together.

3. *Dalrymple's Sign* (Cooper-Swanzy).—This consists of retraction of the upper eyelid so that there is an unnatural degree of separation between the margins of the two lids. The widening of the palpebral fissure produces the peculiar stare which is present in the subjects of exophthalmic goiter, and which has been compared to a similar appearance produced by the action of cocain. This sign and that of von Graefe are probably due to irritation of the sympathetic supplying the superior tarsal muscle.

4. *Moebius' Sign.*—This consists in an imperfect power, or in an entire absence, of convergence, and may be sought for in the usual manner (see page 58). Suker has noted deficient complementary fixation in lateral ocular rotations in exophthalmic goiter.

Changes in the Cornea.—The exposure to which the eye is subject and also the paralysis of the nervous supply may cause drying of the epithelium of the cornea, and ulceration of so violent a type as to produce destruction of the eye. New vessels may develop in the lower part of the cornea on account of its exposure through the widened palpebral fissure. These corneal changes occur in severe types of the disease where protrusion of the eyeballs is conspicuous.

Ophthalmoscopic Changes.—These are not commonly present to any great degree except in so far as a change in the size of the retinal vessels is concerned. The arteries may be dilated and assume a caliber larger than normal and equal to that of the veins. Spontaneous arterial pulsation is frequently present (Becker). Alterations in the optic nerve and in the general fundus are not usually found, and there are no changes in the eye-grounds characteristic of the disease.

Nature of the Disease.—The cause of exophthalmic goiter is not known; but there is little doubt that the symptoms of this disease depend upon a disturbed function of the thyroid gland, whereby there is excessive internal secretion from it, or entrance into the general system of more of its active principle than is normal (Hare).

Treatment.—For the general treatment of exophthalmic goiter the student is referred to the text-books on general medicine, surgery, and neurology. Sympathectomy has been practised for the relief of exophthalmos. Partial thyroidectomy is usually performed and the operation meets with great success. If ulceration of the cornea occurs the usual treatment is indicated. To prevent exposure of the cornea, the widened palpebral fissure may be narrowed by the operation of tarsorrhaphy (see Fig. 306). Sometimes the proptosis is so great that the eyeball is luxated in the sense that it slips forward between the lid borders; this condition may be bilateral and the cornea may be ulcerated (W. R. Parker). Foster Moore has succeeded in reducing exophthalmos by removing a quantity of edematous fat, to which and to edema of the muscles he attributes the proptosis.

Affections or Diseases of the Accessory Sinuses.—In discussing tumors of the orbit it was noted that growths from the frontal sinuses, the sphenoid, the ethmoid cells, and the antrum may encroach upon the orbit. The limits of this book do not permit a full consideration of this subject, for which the student must turn to special treatises. In addition to the morbid growths there remain to be briefly considered:

1. **Disease of the Frontal Sinus.**—This is most often a distention of the frontal sinus by mucus (*mucocele*) or pus (*empyema*). Abscess has been attributed to postnasal catarrh, syphilis, tuberculosis, and periostitis, and is due to the stoppage of the normal outlet, thus causing the accumulation of secretion until the sinus becomes filled, its walls distended and thin, and a tumor presents, usually at the upper and inner angle of the orbit. It may occur under the influence of erysipelas, acute infectious diseases, and epidemic influenza. Sensitiveness on pressure over the frontal bones and frontal headache or supra-orbital pain are common and somewhat characteristic symptoms, and are especially marked in *acute* frontal sinusitis. The protrusion may cause displacement of the eyeball downward and outward and diplopia, and the pressure upon the lacrimal sac, epiphora. Coryza and purulent discharge from the nostril may be present. According to Bull, if a dense, hard swelling appears at the upper and inner angle of the orbit, which is slow in growth and painless, an osteoma of the sinus is almost

certainly present. In rare instances the abscess in the sinus is bilateral. The *x*-rays should be used to establish a diagnosis between osteoma and mucocele of the sinus. A stereoscopic radiogram is essen-



FIG. 282.—Acute frontal and ethmoid sinusitis. Notice the edema of the lid and upper and inner portion of the orbit (from a patient in the University Hospital).



FIG. 283.—Introduction of drainage-tube after evacuation of abscess caused by ethmoiditis. (A patient in the Philadelphia Polyclinic Hospital.)

tial in the study of accessory sinus disease, and careful *transillumination* of the region is required (see also Ophthalmodiaphanoscopy, page 119).



FIG. 284.—Ethmoiditis (from a patient in the University Hospital).

The *chronic* variety of the disease may occur at any age except before the sixth year, because the sinus is not much developed until after

that time of life. It is most frequent between twenty-five and thirty, and commoner in men than in women.

The treatment consists in opening the abscess and washing out the sinus with a bichlorid solution. The incision may be made immediately beneath the superior orbital arch, directly outward, so that the bony wall of the sinus, which is here very thin, may be easily opened, if it has not already perforated. A. Knapp prefers that the external incision shall pass along the upper orbital border midway between the eyebrow and the bony orbital margin, and next along the inner wall and side of the nose to the floor of the orbit. This is better, in his opinion, than the Killian incision through the eyebrow. After the wall of the sinus has been perforated the contents of the cavity should be carefully removed; often polypoid growths are present. The communication between the sinus and the nose should then be re-established, and a drainage-tube passed from the orbit, through the opening, into the nose, or a gauze drain may be passed from without into the sinus at its nasal angle (Knapp).



FIG. 285.—Purulent disease of ethmoid and frontal sinus, with fistulous opening at inner angle of orbit (from a patient in the University Hospital).

2. **Disease of the Ethmoid.**—A common disease of the ethmoid cells is caused by a retention of secretion—that is, adopting H. Knapp's phraseology, a *retention-cyst* develops. In these circumstances the growth appears at the upper and inner angle of the orbit, above and behind the internal canthal ligament, and displaces the eyeball downward and outward. It may not be possible to differentiate this *mucocele* from an exostosis until an exploratory incision is made. With ethmoiditis there may also be tumefaction, especially of the inner third of the lid, imperfect movement of the eyeball with diplopia, severe neuralgic pain, and profuse lacrimation. The last-named symptom may cause the affection to be mistaken for dacryocystitis. In purulent disease of the ethmoid cells the natural escape for the pus is into the nasal cavity, where it can be seen beneath the middle turbinated body, or between this structure and the septa; but this is by no means its invariable course. In a large number of cases pus escapes through

the os planum into the orbital cavity, giving rise to exophthalmos and *orbital abscess*. The purulent collection may be evacuated by a free incision, so placed as to expose the os planum of the ethmoid. After all necrotic and carious tissue is removed, an opening should be forced into the nose. Through it a drainage-tube should be passed, by means of which the cavities can be frequently cleansed with a bichlorid or other antiseptic solution. Often, after partial or complete removal of the middle turbinate bone, the approach can be through the nose and drainage, aided by suction, secured in this manner. Many other procedures are available in the surgical treatment of ethmoiditis, for the description of which the student should consult works on paranasal sinus surgery. Among these H. P. Mosher's operation is particularly valuable.



FIG. 286.—Sarcoma of the orbit and postnasal space (from a patient under the care Dr. Wm. Zentmayer).



FIG. 287.—Exophthalmos from tumor of antrum which involved the orbit (from a patient in the Jefferson Hospital under the care of Dr. J. Chalmers DaCosta).

Fistula of the orbit, presenting above the internal canthal ligament, may be due to disease of the frontal sinus or of the ethmoid, and particularly to disease of the lacrimal division of the anterior ethmoid cells. Cases of this character are often mistaken for lacrimal disease, and, in fact, they present some of the characteristics of the so-called prelacrimal sac abscess. A cure may be effected by forcing with a strong probe, as Gruening suggested, an opening through the base of the fistula into the nasal cavity, thus facilitating drainage through the nose, or, better, by free exploration of the affected sinuses by means of a Killian or Knapp procedure.

3. **Disease of the Sphenoid Sinus.**—Empyema of the sphenoid sinus may exist alone, or more often in association with suppuration

in the ethmoid cells, and may appear in an *acute* or *chronic* form. A diagnosis can often be made by catheterization of the sinus. It is of particular ophthalmologic interest on account of the intimate relation between the walls of the sphenoid cavity and the optic nerve (the optic nerve may even be free in the sphenoid sinus [Onodi]), and an almost necessary symptom is some form of optic neuritis, either retrobulbar or localized in the nerve-head itself. Optic-nerve disease should always induce the surgeon to take the sphenoid and ethmoid sinus into serious account (see also page 677).

In any case of suspected sinus disease a *stereoscopic x-ray* plate should be prepared; this frequently will give accurate information with respect to the size, condition, and the contents of the accessory nasal sinuses.

Other diseases of this region are *polypi*, *osteomas*, and *hyperostoses*.

4. **Disease of the Antrum.**—Empyema of the antrum is not an uncommon affection, and although it does not belong to the domain of ophthalmology, it is sometimes accompanied by marked ocular signs. In addition to the pain located in the cheek, frequently periodic in character, together with the escape of pus from the antrum, there may be a marked edema of the lids, which, if the disease is of long standing, assumes a positively brawny consistency. Edema of the lids may develop when only a few drops of pus are present in the cavity of the antrum. There may also be chemosis of the conjunctiva and some edema of the optic nerve and overfilling of the retinal veins. A persistent edema of the eyelids not otherwise explained should direct the surgeon's attention to the antral cavity. A certain number of cases of lacrimal disease, for example, ordinary forms of dacryocystitis, are connected with antral affections.

Growths in the antrum—*sarcoma*, *fibroma*, and *polypi*—may involve the orbit and produce exophthalmos, or more often displacement of the eyeball upward and outward.

The Ocular Complications of Diseases of the Nasal Accessory Sinuses.—The relation of diseases of the nasal accessory sinuses to diseases of the eye has been referred to in preceding pages in the description of various ocular lesions, notably those which occur in the optic nerve, but for convenience of reference they are redescribed in the following paragraphs:

(1) *Lids and Conjunctiva.*—Edema of the lid is a common symptom of frontal, antral, and ethmoidal sinus disease, either the ordinary variety, or else a recurring painful form, fugitive in character and associated with violent headache. Watering of the eye, conjunctival congestion, distinct catarrhal conjunctivitis, and deep-seated scleral congestions, sometimes fugacious, and often accompanied by intense headache, ocular pain and slight edema of the corneal epithelium, have been noted as frequent symptoms of sinusitis, especially in its acute or early stages.

(2) *The Cornea and Uveal Tract.*—Keratitis, corneal ulcers, iritis, uveitis, choroiditis, and vitreous opacities may be due to sinus disease. A special form of cyclitis with vitreous opacities, which seems to be

due to nasal accessory sinus disease, is described by Kuhnt. (See also page 364.)

(3) *Retina and Optic Nerve.*—The most important group of ocular complications of paranasal sinus disease are those in which there is sinusitis without external signs of orbital inflammation, but in which there are optic neuritis, neuroretinitis, retinal thrombosis, and phlebitis, or in which, without marked ophthalmoscopic changes, there is a central scotoma. In some cases a typical acute retrobulbar neuritis arises, with all of the symptoms which have been detailed on page 562, while in others the retrobulbar neuritis manifests its presence by a relative central scotoma, with intact outlines of the visual field; later the scotoma becomes absolute and the field of vision contracts. The scotoma may be unilateral, the more usual condition, or bilateral, and

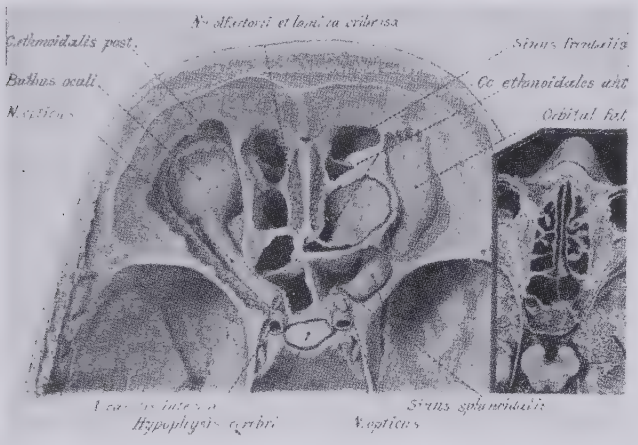


Fig. 288.—Exposure of the paranasal sinuses, orbital tissues, eyeball, and optic nerves from the intracranial aspect. Particularly note the relationship of the left optic nerve to the posterior ethmoidal cells and the sphenoidal sinuses. The right optic nerve pushes the lateral wall of the sphenoidal sinus into a conspicuous mound-like relief. (J. Parsons Schaeffer, in Transactions of the Section on Ophthalmology, Amer. Med. Assoc., 1921.)

most frequently depends upon disease of the posterior ethmoidal cells or of the sphenoid sinus. Occasionally the scotoma assumes a circular or annular shape. The investigations of Onodi and J. Parsons Schaeffer have shown that the optic nerve often is in close relation with these posterior ethmoidal cells, and that the thinness of the intervening wall renders involvement easy, even easier, it is probable, than in the case of the sphenoid, which anatomically may approach the nerve and form the inner wall, or the lower and inner wall, of the optic canal. According to Birch-Hirschfeld, the nerve lesions consist in edema, swelling and proliferation of the glia cells, and destruction of the nerve-fibers. These he attributes to venous stasis and also to toxic agencies. Compression of the optic nerve in the canal, or perineuritis (Hajek) and extension of disease through the intimately related soft tissues of the sinuses, orbit and optic canal (Gradle) have been advanced as factors in the pro-

duction of central scotomas. Bordley concludes that their development depends upon a dual cause—mechanical pressure and toxemia of the papillomacular bundle either from stasis or extension. In a certain number of cases the ophthalmoscope reveals the usual picture of optic neuritis, or papillitis, with central scotoma, especially, as in a case recorded by A. Knapp, if the anterior ethmoidal cells are infected, and the author has seen elaborate optic neuritis followed by optic-nerve atrophy, with extensive disease of the ethmoid, frontal, and sphenoid sinus, and sphenoid-sinus disease in which the scotoma assumed the form of the so-called hemianopic paracentral scotoma. The visual fields in disease of the sphenoid sinus may exhibit alterations analogous to those in affections of the pituitary body, for example, bitemporal hemianopsia. Annular scotoma has also been observed. If the cause of these optic nerve complications is not recognized and speedily removed, either by suitable intranasal drainage, with or without operation, blindness from optic-nerve atrophy is likely to result. An important symptom of posterior accessory sinus disease, described by J. Van der Hoeve, is *enlargement of the blind-spot* for white and colors. This observation has been confirmed by de Kleijn, the author, and a number of others who have investigated the subject. The enlargement of the blind-spot is attributed by Van der Hoeve to involvement of the peripapillary bundle, which may be the first portion affected in retrobulbar neuritis. If no other cause for such increase in the size of the blind-spot can be found, Van der Hoeve considers it to be a symptom which justifies operation on the affected sinus. Bordley found among 102 patients with disease of the posterior ethmoid cells and of the sphenoid enlargement of the blind-spot in 31 per cent. of the cases. This enlargement was more frequently associated with acute sinusitis than with subacute and chronic forms of the disease.

(4) *Orbit and Lacrimal Region.*—A mucocele of the ethmoid or frontal sinus may cause mechanical displacement of the orbital contents and exophthalmos, and Birch-Hirschfeld's investigations have shown that nearly 60 per cent. of the cases of orbital inflammation which he has analyzed were due to accessory sinus inflammation. The infection may cause a periostitis over the floor of the frontal sinus or over the os planum, and bring about exophthalmos. This may disappear as the result of treatment, or a *subperiosteal abscess* may develop, which remains encapsulated, or which, by extension, may perforate the skin of the eyelid, leaving an *orbital fistula.* Such periosteal orbital abscesses are frequent in children, the infection being transmitted by the ethmoid labyrinth (A. Knapp). Reber concluded from his studies that infection may reach the orbit (*a*) by direct continuity, (*b*) by way of the venous circulation, and (*c*) by way of the lymphatics. The last-named route has not been positively demonstrated.

Finally, there may be involvement of the orbital structures themselves, resulting in *cellulitis* or *abscess*, either with or without optic nerve inflammation. Extension of antral disease into the orbit is less common and rarely occurs, according to A. Knapp, except through the

intermediation of the ethmoidal cells, but both with antral and with ethmoidal infection the symptoms of dacryocystitis may appear, and not infrequently the mistake is made of treating as a dacryocystitis a manifestation of sinusitis.

Other complications which have been recorded are glaucoma and detachment of the retina. The former affection does not occur from sinus disease unless the eye is predisposed to increased intra-ocular tension. Intense neuralgia, both ciliary and postocular, stubborn asthenopia, and contraction of the visual field have been attributed to the same cause, dependent, according to Kuhnt, on absorption of toxins from the purulent processes in the sinuses.

Palsy of exterior ocular muscles, notably of the superior oblique, may be caused by sinus disease. The author has observed isolated palsy of the internus due to ethmoiditis, and certain exterior ocular muscle palsies formerly attributed to rheumatism are doubtless due to sinusitis.

Evidently in the presence of any of these conditions, notably persistent or recurring edema of the lids, fugitive episcleral congestion, retrobulbar neuritis, both acute and chronic, optic neuritis, unexplained failure of vision with central and other forms of scotomas, and stubborn ciliary neuralgia with persistent asthenopia, expert examination of the sinuses is demanded, not only by all of the means at the disposal of rhinologists, but notably with the aid of the *x*-rays, and, in some cases, even if the results of ordinary examination are negative, especially in the presence of the optic nerve complications, exploratory orbital incisions and investigation of the sinuses through them are justified. The diagnostic value of *Van der Hoeve's scotoma* has been explained (see page 678).

Although the ocular manifestations of sinus disease are often marked, it should be remembered, as Sattler points out, that excessive dilatation of the pneumatic sinuses of the skull may pursue an entirely latent course and cause no very decided eye symptoms.

Injuries to the Orbit.—These include fracture of its bony walls, penetrating wounds, the lodgment of foreign bodies, and contusions. The effects of an injury of the orbit depend very much upon the character of the wound and the missile which has produced it. Should the fracture be so placed that a communication between the contents of the orbit and neighboring cavities is effected, *emphysema* takes place, recognized by palpation (see page 203). If air accumulates behind the eyeball there may be exophthalmos. Violent contusion of the eyeball, unless the globe is ruptured, may produce *emphysema of the orbit* by pushing the fat against the orbital plate of the ethmoid and establishing a communication with its cells and the orbital contents. The injury may lead to a phlegmonous inflammation, to hemorrhage within the tissues, and to loss of sight because of rupture of the eyeball or injury of the optic nerve. The development of optic-nerve atrophy after injury, evident to the ophthalmoscope, may be delayed for several weeks. There are likely to be, according to the circumstances, exophthalmos, displacement of the eyeball, and diplopia. In warfare, as

exemplified during the recent war, orbital injuries from bullets, fragments of shrapnel, are common and cause various degrees of damage—fracture of the walls, severance of the optic nerve, gross concussion of the eye, or its rupture or disintegration. Fragments of metal may penetrate the globe, pass through, and be buried in the surrounding orbital tissues. So, too, bullets may enter the orbit and lodge in the adjacent sinuses or in the cranial cavity. A complication of many of these orbital injuries on account of the exophthalmos and injury of the orbital nerves may be neuroparalytic keratitis (page 292). Other complications are hemorrhage, which, if excessive, increases the proptosis, orbital cellulitis and *gas bacillus infection* (*Bacillus aërogenes capsulatus*). Air may escape into the cellular tissues of the orbit and produce emphysema which is detected by a crackling sound when the eye is pressed backward.

Treatment.—After a penetrating wound a careful search for a foreign body should be made and if reasonably accessible it should be removed either through the channel of entrance or through a new passage. In a number of instances extraordinary foreign bodies have been found in the orbit, and, curiously enough, very remarkable toleration of the presence of such bodies. If the penetrating wound has cut off the attachment of one of the ocular muscles and the patient is seen soon enough, an endeavor should be made to suture the detached ends. In cases of excessive hemorrhage within the orbit it may be necessary to make an incision and remove the escaped blood. *x*-Ray examination naturally furnishes a means of detecting foreign bodies in the orbit, the position and character of the fracture, and whether the foreign body remains within the orbit or has passed beyond its bounds. A foreign body, for example, a shot or small bullet, if unassociated with infection and not easily removable, had better be allowed to remain than to make injudicious exploration. In some cases of deeply seated foreign body resection of the orbital wall has been the means of securing it. Removal of the foreign body in the presence of suppurative orbital cellulitis followed by free drainage and, in severe cases, Carrel's tubes, is recommended by Greenwood. Metallic foreign bodies can sometimes be removed with a magnet.

Hemorrhage Into the Orbit.—In addition to the orbital hemorrhages caused by injury which have been referred to, or which may follow operation; *spontaneous hemorrhages* may occur. They have been observed in children with scurvy, as the result of arteriosclerosis in old people, in whooping-cough and hemophilia. Cysts following hemorrhage have been described. Arteriosclerotic orbital hemorrhage may manifest itself in a *recurring* form, and the hemorrhage occasioned by scorbutus is often associated with pronounced ecchymosis of the lids and subconjunctival hemorrhage and exophthalmos.

Dislocation of the Eyeball.—The eyeball may be dislocated between the lids, which are contracted behind it. It is a rare form of injury. An eyeball may purposely be pried from its socket by means of a thumb thrust into the orbit from the outer side. Luxation of the

globe as a self-inflicted injury has been observed among the insane. The result of such an accident may be laceration of the optic nerve and destruction of sight. In other instances the vision has remained unaffected. In certain cases of exophthalmos it is possible to produce this dislocation by pressure upon the globe with the thumbs, the relaxed muscles permitting the eyeball to protrude between the lids. The eye should be replaced and bandaged; it may be necessary to divide the external commissure (see also page 672).

Enophthalmos, or retraction of the eyeball, occurs both as an idiopathic and a traumatic affection. Enophthalmos the result of exhausting diseases is more apparent than real, but a true sinking of the globe, producing an appearance not unlike that caused by a badly fitting artificial eye (Nieden), may follow a traumatism in the neighborhood of the orbit (Fig. 289). Enophthalmos, miosis, slight ptosis, and unilateral sweating have been noted in babies after prolonged instrumental labor (Mayou).



Fig. 289.—Traumatic enophthalmos, patient looking straight forward sunken appearance, resembling a badly fitting artificial eye, well shown.

Retraction of the eyeball may immediately follow the injury or be delayed for days or even months. According to the conditions which are present, it has been ascribed to paralysis of Müller's orbital muscle from lesion of the sympathetic (Schapringer); to atrophy of the retrobulbar cellular tissue caused by trophic nerve disturbance (Beer); to fracture and depression of the orbital bones with cicatricial adhesion or contraction; and to injury of Tenon's capsule and the check ligaments (W. J. Shoemaker). It may be associated with palsy of the inferior oblique (Fuchs, Sachs).

Exophthalmos caused by paralysis of the ocular muscles, tenotomies for the relief of strabismus, Graves' disease, nephritis, orbital disease, orbital growths, affections of the nasal accessory sinuses, and thrombosis of the cavernous sinus has been referred to. It may also occur as the result of irritation of the cervical sympathetic under the influence of certain poisons, notably thyroid extract and paraphenylendiamin, in acromegaly, myelitis, and certain tumors of the brain, notably those which are situated in the neighborhood of the third ventricle and in the middle fossa of the skull. Osteomyelitis of the superior maxilla in infants may cause exophthalmos (E. Marx). (See also Proptosis, page 95.)

Intermittent exophthalmos is a rare affection, about 60 cases being on record. The author encountered one example of this affection in an analysis of over 100,000 cases of ocular disorders during the late war. The characteristic symptoms are a more or less rapid, steadily for-

ward movement of one eye when the head is placed in a dependent position, or when the flow of blood from the head to the trunk is impeded to any extent. In other circumstances the eye usually presents a normal appearance, or, on the subsidence of the exophthalmos, there may be a slight enophthalmos. According to Birch-Hirschfeld, intermittent exophthalmos depends upon a varix of the orbital veins, the origin of which may be congenital, although usually the venous stasis does not take place until later in life, and occurs under the influence of the mechanical factors to which reference has been made.

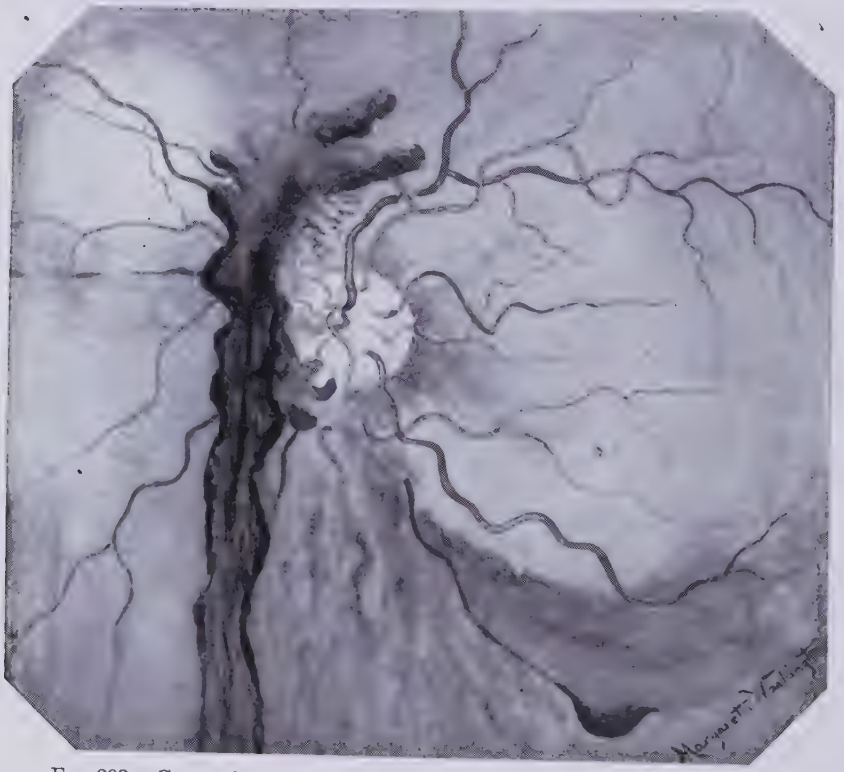


FIG. 290.—Concussioned fundus (from a patient in the University Hospital).

Contusion and Concussion of the Eyeball.—References to some of the effects of contusion and concussion of the eyeball as the result of the violent impact of a blunt object, for example, a flying ball or cork, a clenched hand, etc., have been made in the descriptions of rupture of the sclera (page 327), iridodialysis (page 357), concussion cataract (page 460), rupture of the choroid and holes in the macula (page 409), detachment of the retina (page 513), and commotio retinæ (page 530). It may be convenient to summarize, in addition to these effects of concussion and contusion, their relation to the eye as seen in *warfare*, particularly during the recent war.

In general terms the lesions are caused *directly* by a blow, or sudden forceful pressure on the eyeball, behind, from the side, or tangentially; or *indirectly*, by the transmission of concussion or shock.

Contact lesions are caused, for example, by a missile which grazes the globe but does not rupture it, or by a fragment or portion of a fractured orbital wall or floor or roof, thrust harshly against the eyeball.

Concussion lesions are caused: (*a*) by concussion at a distance, for instance, by violent displacement of air by the explosion of a shell (Lagrange); (*b*) by transmission of concussion or shock through the bony facial structures and, moreover, not only through those near the eye, to wit, the malar bones and orbital margins, but through the superior maxilla (especially if the missile passes through the antrum), and the inferior maxilla; and (*c*) by slight blows on the anterior part of the eye, the concussion being transmitted through the transparent media to the posterior pole (Lagrange).

The lesions as usually described may be summarized thus: (1) Lesions by concussion, (2) lesions by impact, and (3) combined lesions, *i. e.*, both by concussion and impact, the lesions being in front of or adjacent to the spot of contact, and also immediately opposite to the site of impact; or, in another sense, as lesions which are not associated with and lesions which are associated with fracture or perforation of the orbit (passage of a missile through it).

The character and degree of the visual depreciation depends upon the extent, situation, and age of the lesions.

The visual field changes depend upon the location of the lesion and its depth and character. Sir William T. Lister states that "lesions above or below the horizontal plane caused a defect in the field out of proportion to the local disturbance, a 'distribution defect' being found in addition to the local defect due to the lesions." This is due to the fact that not only was the spot struck damaged, but also nerve-fibers in the immediate vicinity which were passing on to a more peripheral portion of the retina. This distribution defect is fan-shaped, the expanded portion being peripheral and the nearer the lesion is to the disk, the greater is the blind sector, and vice versa. When the lesion occurs in the horizontal plane no "distribution defect" is found, as the fibers supplying the retina in the horizontal line arch around from the disk to their destination, and therefore these lesions can only involve the nerve-fibers at their terminations. Scotomas of various shapes may interpret the macular and paramacular alterations.

Marked reduction of intra-ocular tension (*hypotony*) is common in many of these cases. The statement that lowered eyeball tension is an important sign of perforating scleral wounds, and especially of diagnostic import when, for example, a small penetrating wound of the sclera is covered with tumid, and it may be swollen, conjunctiva, must not be taken unreservedly in view of the many observations in this war. Lesions of the inner eye by concussion from a distance, by concussion transmitted through the bony facial structures and following blows on the eye, by preference are located in the macula and paramacular area; impact lesions are equatorial and always adjacent to the site of contact; a contact lesion may spread toward the center; posterior pole and equatorial contact lesions may approach and join each other; missiles traversing posterior to the bulbus and radiating fractures of the orbital vault are responsible for most of the direct optic-nerve injuries. Whether it is safe to say that impact lesions are always, or almost always, retino-choroidal and concussion lesions choroidal, as Lagrange contends, it would seem is not settled.

Unquestionably the difference between concussion changes of the fundus encountered in civil and military practice depends, as Lister points out, in greatest measure upon the fact that in ordinary circumstances the blow is delivered by a comparatively slow-moving object, while in warfare the missile passing through the orbit moves rapidly.

CHAPTER XXI

OPERATIONS

Ophthalmic surgeons, in so far as the preparation of their hands, gowns, operating-rooms, and surroundings is concerned, naturally follow the strict rules of modern surgery.

Preparation of the Skin of the Region of Operation.—The skin should be treated first with soap and water, then with alcohol, and finally with corrosive sublimate (1 : 2000). These irritating substances must not enter the conjunctival sac, but the face, surface of the closed lids, eyebrows, brow, and scalp should be thus prepared. The ciliary margins should be carefully cleansed with soap and water, followed by bichlorid of mercury (1 : 5000). The parts should be kept covered with a compress of lint soaked in the bichlorid solution until the operation begins. In place of this preparation the eyelids may be cleansed with benzine on a cotton swab, to be followed by a thorough washing with fluid neutral soap. This is the practice in Fuchs' clinic.

The preparation of the conjunctival sac depends upon the nature of the operation (see page 761).

Preparation of the Instruments.—All coarse instruments, such as hooks, scissors, etc., should be boiled for at least ten minutes in the usual manner in a sterilizer.

Sharp instruments—cataract knives, keratomes, cystotomes, etc.—must be cleansed with great caution lest damage be done to their edges. First the edge of the instrument is inspected with a magnifying glass, next the instrument, wrapped in cotton, is put into boiling water for five minutes, and from this transferred to a dish containing absolute alcohol, carefully wiped with the cotton saturated in the alcohol, and finally placed in a tray of sterile water. Just before the operation begins, it is removed from the water, thoroughly dried, laid upon a layer of sterile gauze, and covered with another layer of the same material. As boiling is likely to spoil the edges of sharp instruments, Stroschein believes that it is sufficient to rub them with cotton-wool soaked in a mixture of equal parts of alcohol and ether, and subsequently to wash them in a 5 per cent. solution of carbolic acid.

Dressings.—These must be modified according to circumstances. In plastic operations about the lids the ordinary dressings—that is to say, steam- or heat-sterilized gauze—may be applied, held in place with a sterile gauze roller (see also page 674). Iodoform gauze is occasionally useful in packing the orbit after evisceration, although ordinary sterile gauze yields equally satisfactory results.

If a wet dressing is desired, the fabric may be soaked in bichlorid solution (1 : 5000), saturated boric acid solution, or in a physiologic salt

solution which has been sterilized by boiling, the last preparation being especially valuable if skin-grafting has been employed. Bits of sterile gauze or tightly packed pledgets of cotton wrung out from a 1 : 5000



FIG. 291.—Figure-of-8 of one eye.

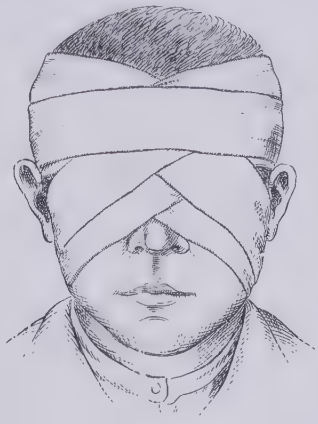


FIG. 292.—Figure-of-8 of both eyes.

bichlorid solution are useful for removing blood, etc., from the area of operation. The various dressings used after cataract extraction, iridectomy, etc., will be described in another section (see page 763).

Either a single or double gauze bandage may be employed, or a modification of Liebreich's bandage.

Sutures.—These may be of catgut, horsehair, or of silk. In the author's opinion silk is always the preferable material, and black silk is more satisfactory on account of the ease with which it can be detected when the time comes for its removal.

General Anesthesia.—The indications for general anesthesia in ophthalmic surgery are limited. In children or in nervous adults, and for enucleations, eviscerations, etc., blepharoplastic operations, occasionally in advancements of the muscles, and in most cases of acute glaucoma, general anesthesia is necessary. The surgeon must decide between ether and chloroform.



FIG. 293.—Modified Liebreich's bandage.

The author prefers to use the former, as it is safer than chloroform or the mixture of chloroform, ether, and alcohol. Bromid of ethyl has been recommended. The author has not been favorably impressed with this anesthetic. The practice of beginning an anesthesia

with nitrous oxid, which is to be continued with ether or chloroform, obviously possesses many advantages. Primary inhalation of ethyl-chlorid is commended by some surgeons. With scopolamin-morphin anesthesia, sometimes employed in surgical operations, the author has had no experience, nor has he had experience with intravenous injections of ether. The administration of ether is greatly facilitated by means of various forms of inhalers and vaporizing apparatus.

Local Anesthesia and Analgesia.—1. **Cocain.**—Hydrochlorid of cocain is usually employed in a 2 or 4 per cent. solution. A 10 per cent. solution has been advised in the operation of cureting lupus and similar growths. General anesthesia is more satisfactory. Cocain causes drying and roughening of the corneal epithelium. This may be partly avoided by keeping the lids closed after each instillation. The drug should not be used too freely, or it may, according to Mellinger, prevent closure of the corneal wound. Gelatin disks impregnated with cocain, as recommended by some surgeons, have no advantage over a solution of the drug. For thorough local anesthesia Haab recommends the application of a thin layer of cocain in crystals. Various fungi grow readily in solutions of this alkaloid, and, indeed, in solutions of any of the alkaloids commonly used in ophthalmic practice. A number of methods of sterilization are employed, namely, sterilization by heat, by the addition of an antiseptic (a 1:5000 solution of bichlorid of mercury, 4 per cent. of boric acid, or trikresol, 1 : 1000, as was suggested by Dr. E. A. de Schweinitz, or by the combination of these two methods). The best method, however, is to boil the solution. A number of convenient flasks designed for this purpose are on the market, among the best being those introduced by Dr. Stroschein, of Wurzburg (since improved and modified by Sidler-Huguenin), and the one devised by Llewellyn, of Philadelphia (Fig. 294). The solution is placed in the latter flask and boiled. After the liquid is cool and ready for use, the warmth of the hand causes the fluid to drop from the end of the pipet. If it is desired to preserve the solution after boiling, a portion of one of the antiseptic substances previously mentioned may be added. The statement often made that boiling a solution of cocain destroys its anesthetic action was not confirmed by the investigations of a committee of the American Medical Association (ophthalmic section), but a fresh solution should be prepared just prior to operation. Cocain is the most satisfactory local anesthetic in operations on the eyeball (excepting, perhaps, removal of foreign bodies from the cornea); its anesthesia is equal to, and almost always greater than, that of any other anesthetic, with the possible exception of butyn. From the experimental standpoint the toxicity of cocain exceeds that of other local anesthetics except alypin, phenacain, and beta-eucain, but these effects are negligible in so far as they pertain to in-



Fig. 294.—Flask for sterilizing collyria.

stillation of its solution into the conjunctival sac. Its penetrating effect is enhanced by the addition of a 0.5 per cent. solution of bicarbonate of soda.

2. **Procain** (*novocain*), although inferior to cocain as an ocular anesthetic, should be preferred to it for infiltration anesthesia, being the least toxic of the local anesthetics. For this purpose it is usually employed in 1 or 2 per cent. solution; it may be combined with adrenalin or epinephrin; the addition of alkalies does not increase its efficiency, and it is not disturbed by boiling the solution (see also page 689).

3. **Eucain** may be obtained in the form of hydrochlorid of eucain "A," which in 2 per cent. solution is an efficient anesthetic, but produces disagreeable congestion of the conjunctiva, and in the form of hydrochlorid of eucain "B," which is related to eucain "A," and also to cocain and tropacain. It is not decomposed by boiling, and may be used in 2 per cent. solution. It is, however, very seldom employed in ophthalmic operations at present.

4. **Phenacain** (*Holocain*).—A 2 per cent. solution of this drug causes anesthesia in from fifteen seconds to one minute, which lasts for about ten minutes, preceded by a moderate burning sensation. It is an admirable local anesthetic, and its solution does not enlarge the pupil, does not affect accommodation nor increase intra-ocular tension nor produce desiccation of the cornea; it possesses slight bactericidal properties. It is preferred by many surgeons to cocain in operations on the eyeball, for example, cataract extraction. Its value in corneal ulceration and to prepare the eye for tonometric examination has been described; it is useful for anesthetizing the cornea preparatory to the removal of a foreign body. A mixture of cocain and holocain is also employed and possesses certain advantages. Alkalies and procain solutions are incompatible.

5. **Butyn.**—This local anesthetic, recently introduced, is efficient in 2 per cent. solution. A single instillation will produce surface anesthesia in one minute; for operative work and deeper anesthesia the instillations should be repeated four times at three-minute intervals. Anesthesia is complete in about eight minutes, and lasts about twenty minutes. Its effect is not harmed by boiling the solution, it has no unfavorable influence on the corneal epithelium, and does not dilate the pupil. Its first instillation produces conjunctival hyperemia and smarting; this may be relieved by adding a drop of adrenalin solution. It has been used for operations on the eyeball (cataract extraction, etc.), and also for muscle operations, and although it is said to be more powerful than cocain, it possesses no advantages over this drug in this respect. It is an excellent anesthetic to prepare the cornea for removal of foreign bodies or for tonometric examination. It has been used in infiltration anesthesia (0.5 per cent.—Bulson), and is said to be satisfactory, but experience along this line is as yet insufficient to reach a definite conclusion.

6. **Acoin.**—This drug is related to caffein and theobromin, and, according to Randolph's experiments, is an active local anesthetic in

unirritated eyes in solutions of 1 : 100 and 1 : 300. It has no effect upon accommodation, the size of the pupil, and does not increase intraocular tension or cloud the corneal epithelium. In congested eyes even repeated instillations of acoin do not produce satisfactory anesthesia. It may be used to prevent the pain of subconjunctival injections (see page 722).

7. **Stovain.**—This drug in 4 per cent. solution, dropped on the conjunctiva, causes smarting, burning, and lacrimation, followed by anesthesia, which lasts for about five minutes. It has little or no influence on the pupil and does not cause paresis of accommodation. In aqueous solution it is not altered by boiling, which renders its sterilization convenient. It has also been used as an injection into the tissues to produce local anesthesia.

8. **Alypin.**—This synthetic compound is a glycerin derivative. A 2 per cent. solution instilled into the conjunctival sac causes slight smarting, some dilatation of the superficial vessels, especially those around the cornea, and anesthesia, which is evident in about one minute (Stephenson). It apparently does not dilate the pupil, and is said to have no influence on accommodation. Neither stovain nor alypin possesses any special advantage in practical ophthalmic work.

9. **Dionin** (*Ethylmorphin Hydrochlorid*).—This is a morphin derivative, which produces, a few seconds after its instillation into the conjunctival sac, smarting, burning, stinging, and marked edema of the conjunctiva, especially of that of the bulbar expansion. Occasionally the lid participates in the swelling, and not rarely the "dionin reaction" is severe. Soon the eye establishes immunity, and after a few applications on succeeding days the reaction is little marked and sometimes does not take place. Within twenty minutes the edema of the primary reaction subsides and analgesia appears, which may last for several hours. The drug is a lymphagogue, an analgesic, and probably has some influence in altering and conserving the nutrition of certain tissues, for example, the cornea. Its lymphagogue action is the important therapeutic one. Its indications have been described in the various clinical sections; it is especially valuable in the treatment of certain types of ulcerative and parenchymatous keratitis, iridocyclitis, and glaucoma. The author employs the drug in a solution varying in strength from 1 to 5 per cent., according to the indications, from once to four times *per diem* until immunity is established; the drug is now discontinued for three days; at the expiration of this time a modified reaction will again usually appear. If it is urgent that the drug shall be continued after immunity is established, the strength of the drug is increased, but never beyond 10 per cent. It may also be employed in salve or powder. It is an exceedingly valuable remedy; occasionally it produces serious reaction; rarely it aggravates existing conditions, especially if the patients are the subjects of arteriosclerosis and renal disease. The value of the drug may be enhanced by preceding its application with holocain and following it with adrenalin-chlorid. Dionin, although an analgesic, is not a local anesthetic, and in that sense

does not belong to those drugs which are used to produce insensitiveness of the tissue with which they come in contact.

With **peronin,** which is related to benzol and morphin, and has an anesthetic as well as a miotic action, and which has been advocated in glaucoma, with **yohimbin,** which is a local anesthetic and which has been investigated in this country by Claiborne, and with **nirvanin** and **apothesin,** the author has had no experience; they appear to be of no practical importance.

Infiltration and Subcutaneous Anesthesia.—Schleich's infiltration anesthesia is produced by an *intracutaneous* (not subcutaneous) injection with a hypodermic syringe, or with one specially devised for the purpose, of a 0.2 per cent. solution of sodium chlorid, which is reinforced by the addition of from 1/100 to 1/50 of 1 per cent. of cocain. The fluid injected produces edema, and the anesthesia is strictly limited to the edematous area.

Subcutaneous injections for local anesthesia may contain as their active agent cocain or procain (novocain). Meller's recommendation is a cocain solution (1 per cent.), 8 to 9 parts, to which is added adrenalin (1 : 1000), 1 to 2 parts; the adrenalin content is not allowed to exceed $\frac{2}{10}$ c.c. A precisely similar solution, substituting procain for the cocain in 1 or 2 per cent. strength, usually the latter, is much employed, and if large quantities of the solution are required, is much preferable to the cocain mixture. In the author's clinic it is the custom to use a procain (sometimes a cocain) solution of the strength noted in physiologic salt solution (see also pages 687 and 688).[1]

Siegrist's Method of Local Anesthesia.—The fluid consists of a 1 or 2 per cent. solution of procain (holocain), to which, after sterilization, a few drops of adrenalin (1 : 1000) are added. In enucleation of the eyeball the method is as follows: After the conjunctival sac has been anesthetized in the usual manner, with a curved cannula needle attached to a glass syringe of 2-c.c. capacity, two injections are made into the posterior part of the orbital cavity behind the eyeball, the needle being inserted on the nasal and temporal sides below the horizontal line, so that it shall not pass through the muscles. Four instead of two deep injections may be made, up, down, in and out, 0.75 c.c. of the fluid being used at each point. Because Seidel has found that this method does not always create satisfactory anesthesia, he has modified it in that with a straight needle he injects 1 to 2 c.c. of the novocain-adrenalin solution around the cornea, 4 mm. from its border. Next four deep injections are made, the needle being inserted upward, downward, outward, and inward over the muscular insertions to a point midway between the optic nerve entrance and the optic foramen.

[1] The whole question of local anesthesia is admirably discussed in a report presented by a committee of the American Medical Association (Ophthalmic Section) composed of A. E. Bulson, W. Zentmayer, E. S. Thomson, H. M. Langdon, and J. Whitaker. Their report also contains a résumé of the principal points in Duverger's comprehensive work "L'Anesthesie locale en Ophthalmologie." A special report on butyn by the same committee will be found in the Amer. Jour. Ophth., 1922, v, 57.

Each injection should contain 1 c.c. of the solution. The immediate results are edema of the lids and exophthalmos, which may be reduced by pressure. Elschnig and Lowenstein produce local anesthesia by deep injections in the neighborhood of the ciliary ganglion, the fluid being introduced by means of a long, straight needle (5 cm. long) along the inferior border of the external rectus; it must penetrate at least 4.5 cm. Local anesthesia may be employed in ordinary enucleations, save only that it is not satisfactory in children and nervous persons, nor should it be used if the eyeball is badly shattered or in a state of panophthalmitis. Angus MacGillivray advocates the following solution: procain, 10 grains (0.6 gm.); sodium chlorid, 2 grains (0.13 gm.), potassium sulphate, 1½ grains (0.09 gm.); distilled water, 1 ounce (30 c.c.).

Local Hemostasis.—For the purpose of producing a hemostatic and astringent action the surgeon may employ various preparations of the *suprarenal capsule.* The dried and powdered gland (1 part to 10 parts of water) has been used; other preparations are *atrabilin*, *suprarenin*, and *epinephrin.* The most satisfactory preparation (containing the principle isolated by Takamine) is *adrenalin chlorid.* It is efficacious in a solution of 1 : 10,000, and is active in even weaker solutions; as dispensed the solution is of a strength of 1 : 1000. This preparation is used for controlling hemorrhage during slight operations on the eye, for example, tenotomies, excision of pterygia, etc.; for temporarily blanching a congested conjunctiva, and especially, if it is desired to differentiate the types of injection in the different sets of vessels (see page 50); as an adjuvant to the physiologic action of certain remedies, for example, eserin in glaucoma, atropin in iritis, etc.; to enhance the value of certain subcutaneous injections for the purpose of producing local anesthesia and controlling hemorrhage (see page 689); and, finally, as a therapeutic agent, the indications for which have already been given.

OPERATIONS UPON THE EYELIDS

Epilation of the Eyelashes.—Removal of the lashes is performed with forceps known as *cilium forceps* (Fig. 295).

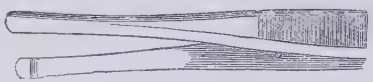


Fig. 295.—Cilium forceps.

The patient being seated in good light, the operator with the fingers of one hand puts the lid upon a stretch, at the same time slightly everting its border. The faulty cilia are firmly seized and pulled out with a quick motion. After those which are readily seen have been removed, search should be made (with a loupe) for others which may have been broken off, leaving small but irritating ends, and for very fine white airs which, owing to their lack of color, may escape detection by the unaided eye.

Removal of a Meibomian Cyst.—This may be removed by a conjunctival incision. A sharp scalpel and small curet are required.

The lid is everted, and the discolored patch marking the position of the chalazion is made prominent. This is incised, and the contents are scraped out thoroughly with the curet. The cavity thus formed fills with blood, the absorption of which may be hastened by the use of hot compresses. This operation may leave a slight linear scar in the conjunctiva (Fig. 297). It is an advantage to continue the incision to the margin of the lid in the line of the duct as recommended by John Dunn.



Fig. 296.—Chalazion curet.

To avoid a scar the lid may be grasped between the thumb and forefinger, and by pressure a drop of the jelly-like contents made to appear at the mouth of the Meibomian duct. A few drops of cocain solution are injected by means of a hypodermic syringe the needle of which is pushed into the tumor along the duct. An incision is now made with a Graefe knife, following the course of the needle. A small curet is introduced, and the contents of the cyst are removed (Agnew-Ray). The subsequent blood-clot undergoes absorption.

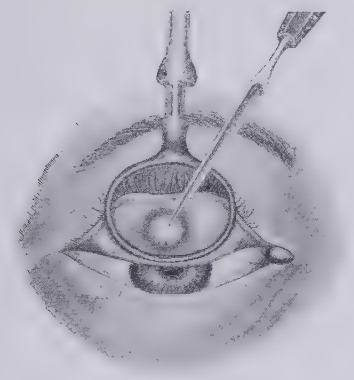


Fig. 297.—Incision of a chalazion (Czermak).

External chalazion should be removed through a skin incision, the lid being steadied in a clamp (Fig. 298), and the cyst dissected from its bed in the ordinary manner.

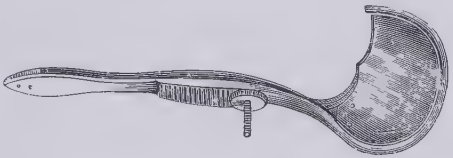


Fig. 298.—Knapp's lid clamp.

Operations for Ptosis.—Before operating for the relief of ptosis the amount of power residing in the levator, or whether it has any activity at all, should be ascertained. The surgeon, standing in front of the patient, firmly depresses the eyebrow with his thumb and requires the patient to open his eyes. Any movement of the lid must be due to the levator, as the pressure on the brow checks the frontalis action; entire failure of lid elevation indicates absence of levator power. If, the frontalis action still being checked and the levator power absent, there is slight elevation of the lid when the eye is rolled upward, it is due to the action of the superior rectus, from which a band passes to the levator tendon.

All operations for the relief of ptosis, and many have been devised, may be gathered into three groups, according to the convenient classification of Grimsdale and Brewerton: (1) Those which shorten the lid or levator; (2) those which utilize the action of the frontalis muscle (3) those which utilize the action of the superior rectus.

The simplest of the first group, namely, an excision of an elliptic

portion of the skin of the lid, is an operation which should not be performed on account of its inefficiency.

Eversbusch's Operation.—The lid is drawn downward and fastened with Knapp's clamp. An incision is now made through the entire width of the lid midway between its margin and the eyebrow, which divides the skin and orbicularis muscle. The edges of the wound are separated for 4 mm. from the underlying tissue above and below, and the tendon, which is thus well exposed, is next included in a loop, with the aid of three double-armed threads passed respectively at the center, the nasal, and the temporal margins. Each needle is now thrust vertically downward between the tarsus and orbicularis, brought out at the free margin of the lid, and securely tied after the wound on the surface of the lid has been closed in the usual manner.

This operation is intended for the relief of imperfect action of the levator and is designed to advance its insertion.

Other operations belonging to this class are advancement of the levator tendon, as designed by Wolff and modified and improved by Elschnig, and excision of a semilunar piece of tarsal cartilage, uniting the edges of the wound with sutures, as advised by Gillet de Grandmont, which is a modification of an operation long ago suggested by Bowman.

To the second group belong a number of subcutaneous thread or wire operations, which act by establishing a contracting cicatrix or by supplying an artificial tendon.

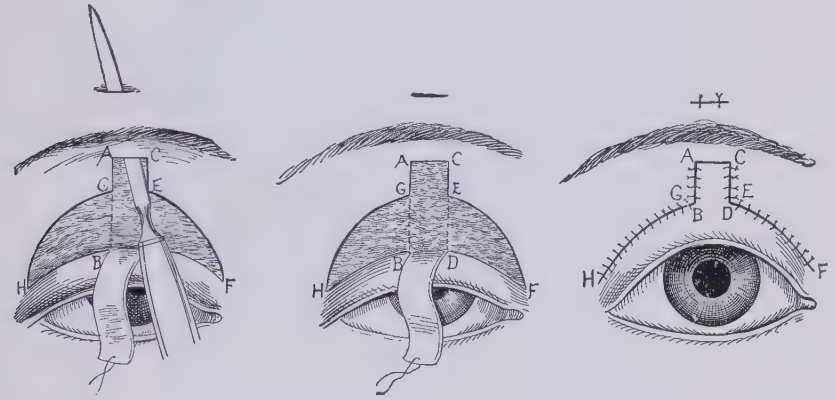
Pagenstecher's Subcutaneous Thread Operation.—A silk suture armed with two needles is provided. One needle is introduced close to the ciliary border and passed subcutaneously for 2 mm. parallel to the ciliary margin. Next the same needle is re-entered at the point of exit and passed between the tarsus and skin and brought out above the brow. The second needle is introduced at the point of entrance of the first and passed upward beneath the skin to the point of exit of the first above the brow. Finally the sutures are tied.

This method establishes a contracting cicatrix and is suited to cases of incomplete ptosis. The operation was modified and elaborated by the late Mr. Mules, who embedded a fine loop of gold wire in the tarsal cartilage, the two ends from which passed out through the frontalis and which remained and acted as an artificial tendon. Worth uses kangaroo-tendon for the same purpose, and Harman "wove-chain" made of fine wire, which is passed subcutaneously from the lid margin to a point below the brow.

A number of operations have been designed to form a union between the skin of the lid and the frontalis muscle, and among these is the well-known *Panas operation*, which consists essentially in the formation of a small cutaneous flap from the lid, which is passed through an incision under the brow and is attached to the fibers of the occipitofrontalis muscle, which have been divided by an incision immediately above the brow, and which has cut through all the tissues down to the periosteum. This operation, once much employed, is now rarely performed, probably because it is apt to produce an unsightly folding of the lid.

J. O. Tansley has designed a combination of the Panas and von Graefe operation, or rather, according to M. L. Foster, a modification and improvement of Hunt's operation, with which the author has had gratifying success:

"Two perpendicular and parallel cuts, *A–B*, *C–D* (Figs. 299–301), $\frac{1}{4}$ inch apart, are made, and extend from the upper orbital margin to within two lines of the upper edge of the lid. These cuts are united at the upper extremity by a horizontal incision, *A–C*, and the ribbon of tissue is dissected up and permitted to drop down upon a wad of cotton lying on the cheek, which is kept moistened with a warm saline solution. Next, a curved cut is made from *H* to *G* and from *E* to *F*, following the crease, which shows the upper limit of the tarsal cartilage, and a straight cut is made from *H* to *B* and from *D* to *F*, parallel to and about two lines distant from the lower border of the lid. The skin and the orbicularis embraced within these cuts are now carefully dissected off, leaving the whole tarsal cartilage denuded of tissue. The cut edges *H–G* and *E–F* are united to the cut edges *H–B* and *D–F*, respectively, by interrupted sutures. Next, a narrow Graefe knife is entered at *A–C*, and passed beneath and brought out upon the forehead just above the eyebrow, and slight



Figs. 299–301.—Tansley-Hunt operation for congenital ptosis.

lateral cuttings are made so as to give room for the passage of the ribbon of skin which has been dissected up at the first stage of the operation. A strong suture placed in the upper edge of this ribbon of skin is used to draw it up into the cut made beneath the eyebrow and bring it out upon the forehead. When it is drawn up sufficiently tight, it is cut off smooth with the forehead and fastened there by two small sutures. Then several sutures are placed from *A* to *G* and *C* to *E*, uniting the edges of the ribbon to the bordering derma." The operation can be readily understood by reference to Figs. 299–301.

The effect of this operation is well shown in the accompanying illustration. Although the tongue of skin as it passes beneath the brow is very evident, in the course of time this appearance subsides and the cosmetic results are good.

Hess's Operation.—This procedure is an elaboration of Pagenstecher's suture operation and achieves excellent results. It is performed as follows:

A horizontal incision is made through the skin of the shaved eyebrow as long as the palpebral fissure and the dissection carried on until the skin is undermined almost to the free border of the lid in its entire length, forming, therefore, a four-cornered pocket. Next three strong double armed black silk sutures are introduced thus: one needle of the first or middle suture is passed through the skin 6 mm. from the lid border and carried in beneath the skin and between it and the orbicularis



FIG. 302.—Ptosis, showing stitches in Tansley-Hunt operation (from a patient in the University Hospital).



FIG. 303.—Complete Congenital Ptosis.



FIG. 304.—Ptosis (Fig. 303)—showing results of Tansley-Hunt operation.

muscle to the line of the first incision; the second needle of the suture is similarly passed, having been introduced about 5 mm. from the point of entrance of the first needle. In like manner at a distance of 1 cm. on each side of the middle suture the two remaining double armed threads are passed. Following this the needles of the middle suture are made to penetrate deeply from the upper edge of the original skin incision close to the periosteum and hence beneath the muscle and brought out a few millimeters above the brow or about 1.5 to 2 cm. from the first incision. The same maneuver is repeated with the lateral sutures, the inner one being inclined slightly toward the median line. The sutures are tied, the thread being drawn tightly over small rolls of surgical gauze or short pieces of narrow drainage-tube. Thus the lid is raised, and the skin, folded on itself, is in the position of the natural fold and establishes a new adhesion to the muscle. A few interrupted sutures close the lips of the original incision. These sutures may be removed at the end of three days, but the other sutures should remain for at least two weeks. To protect the open eye it may be covered with a celluloid shield shaped like a large



FIG. 305.—Result of a Hess operation for ptosis, showing on left side the position of the stitches (from a patient operated upon by Dr. T. B. Holloway).

watch crystal fastened with strips of gauze and collodion or a Ring mask (Fig. 377) into which a square window is cut, to be covered with a layer of gauze, may be fitted over the face. The suture lines may be painted with Whitehead's varnish or with a 5 per cent. solution of iodin.

The operation is indicated when the patient's ability to correct the ptosis by contracting the frontalis muscle is demonstrable; otherwise satisfactory results are not achieved. An occasional complication is ulcer of the cornea; this occurred once in the author's experience; Meller reports a similar experience and Posey has observed a keratitis in these circumstances of unusual severity.

Other operations belonging to this group are the procedure of W. H. Wilder, who folds upon itself the tarso-orbital fascia and establishes a firm adhesion between the fascia and the frontalis muscle, Fergus'

method of attaching a strip of the frontalis to the lid, and Sourdille's modus operandi, by which the levator tendon is fastened to the frontalis.

Finally are those operations which depend for their effect upon a utilization of the action of the superior rectus. Among them the one most frequently employed is that designed by Motais (*Motais' operation*), which consists essentially in attaching a narrow tongue of tissue formed from the center of the tendon of the superior rectus, through an opening in the conjunctival surface of the everted lid, to the upper border of the tarsus, where it is fastened by means of sutures which are brought out through the tarsus and lid skin and tied on the outer side of the lid. While this operation has certain attractive features, it may be followed by temporary diplopia and depression of the eyeball. If the sutures are tied over the conjunctiva, the knot may cause local irritation and even corneal ulceration. If the tendon of the superior rectus is poorly developed, W. T. Shoemaker suggests that the entire tendon of the rectus muscle, in place of a single central strip, shall be fastened to the tarsus in the manner described.

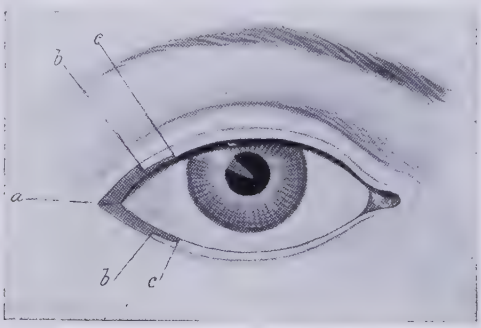


Fig. 306.—Lateral tarsorrhaphy.

After any of these operations, performed with the usual aseptic precautions, the ordinary dressings should be applied and the sutures removed at the end of a week. The method of protecting the cornea has been referred to. The anesthesia may be local or general.

Tarsorrhaphy.—This operation is designed to shorten an abnormally wide palpebral fissure (*lateral* or *angular tarsorrhaphy*) or to close temporarily the lids over the eyeball (*median tarsorrhaphy*). Lateral tarsorrhaphy is performed as follows:

The external commissure is taken between the thumb and index-finger, the fissure of the lids closed to the required extent, and the line of incision marked with an anilin pencil. A horn spatula or shield is now introduced between the lids, and a flap removed from the free margin of each lid near the external commissure; this must contain all the hair-follicles. The breadth of the flap is 1 mm. and the length about 4 mm. To obtain still firmer union the ciliary margin may be denuded for several millimeters beyond the point of removal of the flap, but in this incision the cilia must not be injured. The edges are approximated by silk sutures. Figure 306 explains the steps: *a* indicates the point of union of the two flap wounds behind

the commissure; *b*, *b*, the termination of the flap wounds in the lid margins; and *c*, *c*, the end of the denudation of the ciliary margins.

Median tarsorrhaphy is accomplished by denuding the ciliary margin of the center of each lid for 4 mm., the lashes being untouched, and approximating the denuded edges with a mattress-suture. The eyeball is thus effectually covered, but the cornea can be inspected if the globe is rotated either inward or outward through the narrowed lid interspace on each side of the central attachment.

Angular tarsorrhaphy is indicated in ectropion in order to raise the angle of the lid, and in lagophthalmos and exophthalmos to improve the unsightly appearance and to protect the cornea.

Internal tarsorrhaphy is sometimes required. In these circumstances the internal canthus and the lacrimal canaliculi must be spared. Occasionally it is necessary to combine external and internal tarsorrhaphy, for instance, in cases of extreme exophthalmos. Meller recommends complete closure of the palpebral fissure after extensive corrosion

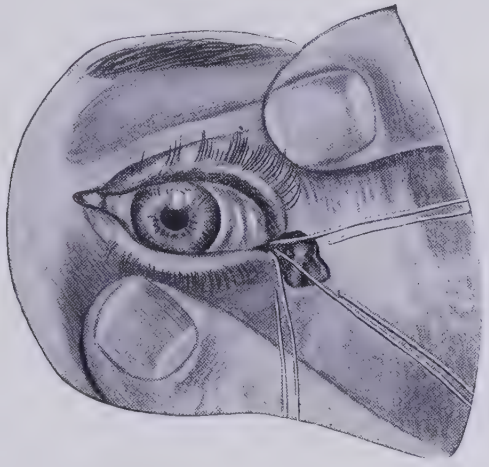


Fig. 307.—Canthoplasty. The stitches ready to be tied (Haab).

of the skin of both upper and lower lids to prevent cicatricial ectropion.

Median tarsorrhaphy is suited to those conditions in which the cornea must be protected, for example, in exposure-keratitis, facial palsy, and to prevent ulceration after removal of the gasserian ganglion (page 293).

Canthoplasty (*Blepharotomy*).—This operation is performed to enlarge an abnormally short palpebral fissure.

One blade of a pair of probe-pointed scissors is introduced behind the external commissure, and the entire thickness of the tissues is divided, making the wound in the skin a little longer than that in the conjunctiva. The wound margins are next separated, and the surgeon loosens the conjunctiva at the apex of the incision and frees it from the underlying tissue. Three sutures are passed, one uniting the extremity of the conjunctival flap to the center of the skin incision, and one suture above and one below, near the angles of the wound. Division of the external canthus without subsequent introduction of sutures is known as *canthotomy*.

Canthoplasty is frequently performed for the relief of the contracted fissure which follows long-standing trachoma and certain types of chronic blepharitis, and also to lessen the tension on flaps in various types of blepharoplasty. Axenfeld recommends *blepharotomy* prior to cararact extraction where the palpebral fissure is extremely narrow.

Operations for Trichiasis.—If only a few hairs are involved, the offending lashes should be extracted with cilium forceps in the manner already described.

Electrolysis, as originally suggested by Michel, of St. Louis, may be performed as follows:

A platinum or iridium needle attached to the negative pole of a constant battery is inserted into the follicle of the lash which is to be removed. A sponge electrode attached to the positive pole is applied to the cheek and the current closed. A drop of froth appears around the needle, which should be kept in place for a few seconds and then withdrawn. The lash is easily removed.



Fig. 308.—Method of making the intermarginal incision (Czermak).

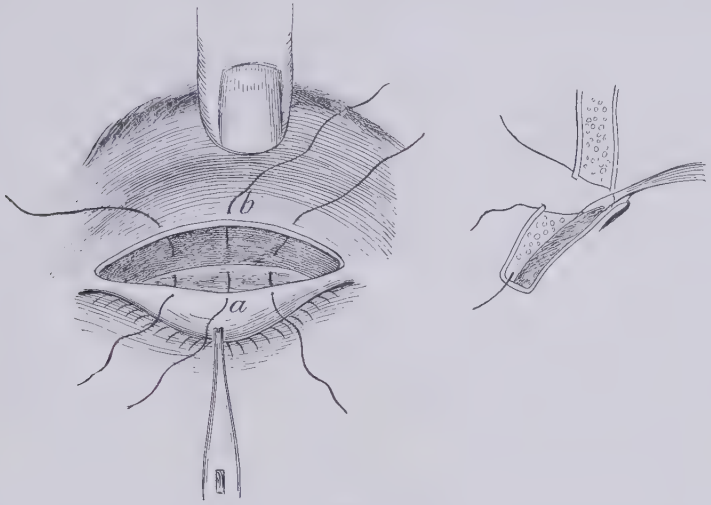
For complete distichiasis some form of transplantation should be employed. The Jaesche-Arlt operation, once often recommended, is not satisfactory, and has been abandoned for more rational procedures.

Double-transplantation operations, or, in other words, the manufacture of an artificial lid border by transplanting a strip of skin to the intermarginal space, have been practised, especially since Spencer Watson's suggestion.

F. C. Hotz designed the following valuable method which the author employs with most satisfactory results:

The lid border is split by the well-known intermarginal incision (Fig. 308), after which a transverse incision is made through the lid skin and the orbicularis muscle just below and parallel with the upper line of the tarsal cartilage. The strip of muscular fibers which covers the upper portion of the cartilage is excised, and the lid skin is united with the upper border of the cartilage by three sutures. Each suture passes through the edge of the lid skin, then through the upper border of the cartilage, and finally through the upper edge of the cutaneous wound (Figs. 309, 310). After the sutures are tied the skin of the lid is drawn upward and fastened

to the upper border of the tarsus. By this means a thorough eversion of the anterior edge of the split-lid-border is effected, and the intermarginal incision is converted into a gaping wound several millimeters in depth. This groove is filled with a skin-graft, long and narrow and somewhat wedge shaped, which preferably is removed from the integument behind the ear. It should be from 1½ to 2 mm. in width, and of a proper length to fill the opening. The graft is spread out, gently pressed into the groove (Fig. 311), and after thorough irrigation with a saline solu-



FIGS. 309 and 310.—Operation of Anagnostakis and Hotz.

tion both eyes are covered with a compress bandage. During the first two weeks the epidermis of the graft is repeatedly shed, and it is advisable to keep the new lid border well lubricated with bichlorid-vaselin (1 : 3000).

Because the fine cutaneous hairs in the transplanted flap sometimes irritate the cornea Van Millingen proposed his *tarsocheiloplastic operation,* in which the intermarginal gap is covered with a strip of mucous membrane taken from the inner surface of the under lip.

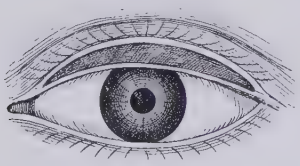


FIG. 311.—Reconstruction of the lid border; skin-graft fills the intermarginal groove (Hotz).

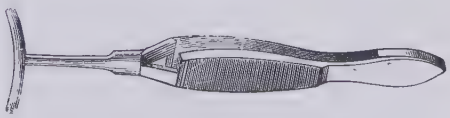


FIG. 312.—Cross-bar entropion forceps.

Operations for Entropion.—Several methods of correcting spasmodic entropion have been referred to on page 198. *Gaillard's suture* is also useful; the skin of the lid is temporarily shortened by means of a fold caught in one or two sutures; the needles being passed from the most prominent part of the entropionized lid through the skin as far as

the lower border of the orbit where the threads which loop the fold of skin are tied over small rolls of gauze. Two folds of this character may be formed at the junction of the inner and middle and of the middle and outer third of the lid. In the *spasmodic entropion* of elderly people the following operation may be performed:

With entropion forceps a strip of skin of suitable width, parallel to the ciliary border of the lid, is pinched up. This strip, together with the subjacent fibers of the orbicularis muscle, is excised. The wound is closed with silk sutures and dressed in the ordinary way. The sutures are removed on the third day.

Instead of excising a horizontal fold of skin, excision of a triangular portion may be performed (von Graefe). A horizontal incision is made in the lid skin about 3 cm. in length and 3 mm. from the ciliary margin. At its center a small triangular flap is formed by two incisions converging below, its length and width being calculated according to the looseness of the tissues. After the flap is excised the margins are freed and brought together with sutures, but no sutures are applied to the horizontal incision. If necessary, the subjacent tarsal cartilage may be removed.

In organic entropion an operation must be performed which will not merely evert the misplaced border, but also alter the curve of the tarsal plate, which usually has become thickened. Two operations will be described:

Burow's Operation.—This operation is designed to relieve entropion of the upper lid following trachoma. It is performed as follows:

The upper lid is thoroughly everted, and the grayish-white scar-line (see page 240) which runs parallel with the margin of the lid is exposed. At the temporal end of this line an incision is made sufficiently large to admit a fine grooved director, which is now pushed to the nasal side of the lid between the skin and the conjunctiva, care being taken that the point of the director is kept well beneath the cicatricial tissue. The tissue thus elevated is divided in its whole length either with a sharp scalpel or with narrow scissors. When the operation is completed a blue line equal in length to the line of incision should appear upon the cutaneous surface of the lid. No dressing is required, or, at most, cold compresses to allay the irritation. The cicatricial contraction which ensues everts the incurved border of the lid.

Although the operation is usually primarily successful, its effects generally do not long remain, and recurrence of the entropion takes place. The operation may be repeated several times.

Hotz-Anagnostakis Operation.—This procedure, as it was practised by Dr. Hotz, is described in his own words, as follows:

"A transverse incision from canthus to canthus is made through skin and subjacent tissues, but instead of being made near and parallel with the free border (as in the former methods) the incision in this operation is to follow the *upper* border of the tarsus. It therefore describes a slight curve beginning and ending at a point about 2 mm. above the canthus, but being 6 to 8 mm. distant from the free border in the center of the lid. While an assistant is holding the edges of the wound well separated, the surgeon lifts up with forceps and excises with scissors a narrow bundle of the muscular fibers which run transversely along the upper border of the tarsus. Next the sutures, which are to include nothing but the cutaneous wound borders and the upper border of the tarsus, are inserted. The first suture is placed in the center of the lid; the curved needle, armed with fine black aseptic silk, is passed through the lower wound border; there taken again in the needle-holder, it is boldly thrust through the upper border of the tarsus, and returned through the

tarso-orbital fascia just above this border; and finally it is carried through the upper wound border (Fig. 309, *a*, *b*). One similar suture is placed at each side of the central one, and these three stitches are usually sufficient for our purpose—to wit, to draw the skin of the eyelid up toward the upper border of the tarsus and establish a firm union between these parts. This artificial union produces a slight tension of the tarsal skin, which, however, is sufficient to relieve any ordinary degree of entropion.

"But when the lids have been badly contracted—when the palpebral aperture has become unnaturally narrow, or the free border of the lid has become entirely merged into the plane of the conjunctiva—these complicated cases require, in addition to the above operation, such surgical measures as canthotomy, the restoration of the free border either by grooving the tarsus or by grafting" (see description, page 699, Fig. 311).

This is a most satisfactory operation, and the results in the author's experience have been excellent. If grooving the cartilage—that is, cutting a groove or narrow gutter along the center of the tarsus from one end to the other—is combined with the Hotz-Anagnostakis operation, the double knife and clamp devised by W. H. Wilder may be employed (Fig. 313). This knife is also most useful for cutting the narrow graft of skin which may be required to restore the lid border and which is applied in the manner already described (see page 699).

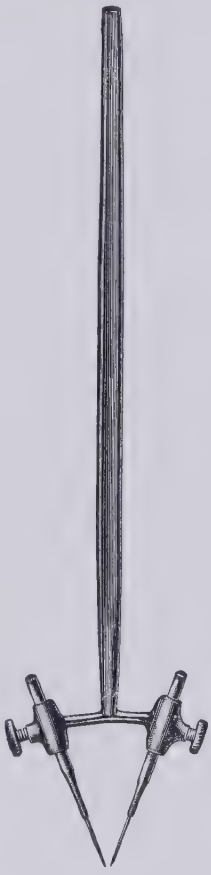


Fig. 313.—Wilder's double knife.

Although the operations for trichiasis and entropion have been separated in the descriptions, it must be remembered that these two conditions are constantly associated, and hence their surgical treatment in most particulars is identical. Many other operations for the relief of these and other lid affections have been devised, but necessarily the author has described only few standard methods, and especially those which have given him personal satisfaction.

Operations for Ectropion.—Certain recommendations for the treatment of *acute* or *spasmodic* ectropion appear on page 199. The condition, however, may persist or constantly recur, usually in so far as the lower lid is concerned. In these circumstances the tarsal conjunctiva presents as a swollen, red roll; the nutrition of the cornea is threatened and a more definite replacement of the lid must be secured, for instance, by the following operative procedure:

Snellen's Suture Operation.—A suture armed with a needle at each end is provided. One needle is entered at the junction of the external and middle third close to the posterior border of the tarsus, and is passed down beneath the skin of the lid to a point at the summit of the lower margin of the orbit, and is there brought

out. The second needle is entered at a point 5 mm. from the first, and with the other end of the thread is carried down close to the first and parallel with it. The two extremities of the suture are tied upon the cheek over a piece of drainage-tube. The same procedure is repeated with a second double-armed suture, the points of entrance being at the junction of the middle and inner third of the conjunctival surface. This operation is suited to cases of spastic ectropion. It has been employed in senile ectropion, but has in these circumstances no valuable permanent effect.

Galvanocautery puncture is recommended by S. Lewis Ziegler for the relief of ectropion and entropion. A lid-clamp is adjusted and the galvanocautery point is pushed through the tarsus and quickly withdrawn. The punctures are made 4 mm. from the lid margin, and separated from each other by an interval of 4 mm. They are made on the conjunctival surface in ectropion, and skin surface in entropion.

For the relief of ectropion associated with relaxation of the tissues, eversion of the conjunctiva which is inflamed and thickened, and an irritating epiphora, as it is often seen in old people (*senile ectropion*), excision of a V-shaped piece of the whole thickness of the lid was at

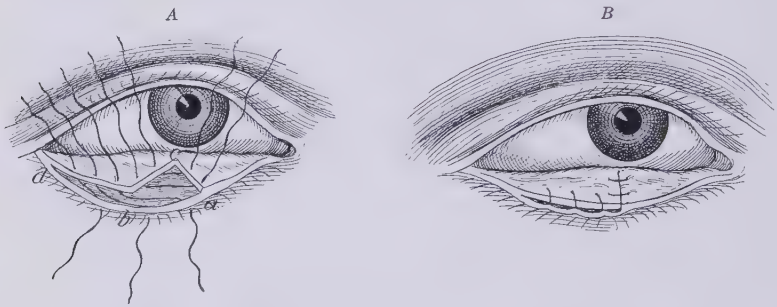


Fig. 314.—*A*, Shortening of lid border after manner of Kuhnt and Müller; *B*, Kuhnt-Müller operation, final stage (Hotz).

one time much practised. This procedure is not at all satisfactory, and one of the operations next described should be performed.

Kuhnt-Müller Operation.—With a broad, triangular knife a deep incision is made into the center of the lid margin, which divides the lid substance into two portions, the one containing the conjunctiva and the tarsus, and the other the soft tissues and the skin. From the first portion a triangular piece is removed by the aid of two incisions which should converge toward the fornix (Fig. 314, *A*, *a–c* and *b–c*). The two portions of the lid are next separated toward the external canthus by carrying the knife from under the margin *b–c* toward *d*. Next, the V-shaped wound of the tarsus is closed with sutures, and the long stretch of the skin margin, *d–a*, is united with the shorter margin, *d–b*, of the tarsus by means of sutures. Their method of application may be understood by examining Fig. 314, *B*. The puckering which occurs after these sutures are tied disappears and the lid margin becomes smooth.

Kuhnt-Szymanowski Operation.—An even more satisfactory operation for the relief of senile ectropion is a combination of the methods of Kuhnt and Szymanowski.

The first step of the operation (Fig. 315) is performed exactly as in the Kuhnt-Müller procedure (Fig. 315). The next step consists in the excision of a tri-

angular piece of the skin at the external canthus in the manner indicated in the diagram (Fig. 316). The base of this triangle should be somewhat longer than the base of the triangular piece which has been excised from the tarsus. A second incision is carried from the canthus downward and slightly outward and should be twice as long as the incision from *a* to *b*. Finally, the two incisions are united in

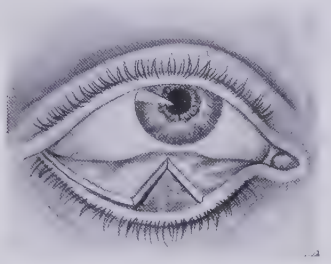


FIG. 315.—Showing the division of the lid into two portions and the excision of the triangle of thickened conjunctiva and tarsus. Kuhnt-Szymanowski's operation (after Meller).

such a manner that the triangular piece of skin can be excised. The skin of the lid is next thoroughly undermined, so that it may be easily drawn outward to cover the defect which has been produced by the excision of the triangular area at the outer canthus. The operation concludes with the insertion of silk sutures in the manner shown in Fig. 317.

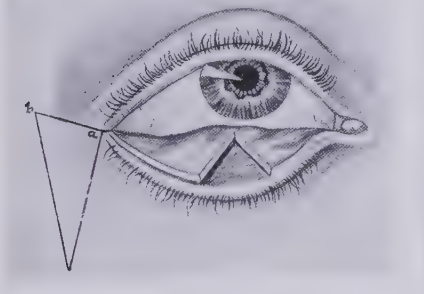


FIG. 316.—Showing the formation of the triangle of skin which is later removed. Kuhnt-Szymanowski's operation (after Meller).

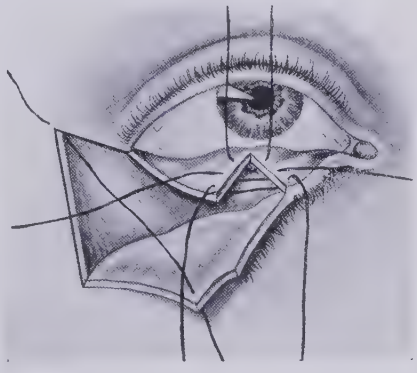


FIG. 317.—Showing the condition after the excision of the triangular piece of skin and the undermining of the lid, which is turned outward. The sutures are in place. Kuhnt-Szymanowski's operation (after Meller).

The operations for the relief of ectropion thus far described are in general terms suited only to those types of this lid deformity unassociated with loss of tissue after injury or removal of growths, scar-tissue, and the results of contracting cicatrices. In the presence of such complications operations of much more formal character and elaborate technic are required.

Operations for Cicatricial Ectropion (*Blepharoplasty*[1]).—Much can be accomplished in the early treatment of wounded and lacerated eyelids and their surroundings, to prevent subsequent deformities and contractions. Scrupulous aseptic cleansing of the injured areas is of paramount importance, and as Gillies maintains, all tissue should be retained the retention of which is feasible; it should be put back into its normal place at as early a date as possible. Sutures must be neatly inserted and with due regard to a coaptation of the lacerated parts in their proper positions.

Before attempting the correction of lid deformities it is important to eliminate sources of infection in their neighborhood; suppuration of the conjunctival mucous membrane by suitable antiseptic irrigation; pus in the lacrimal sac by its excision; and purulent secretion of the ethmoid, frontal, and antral sinuses; and to postpone operative interference until edema and inflammation have subsided and cicatrization is complete.

In general terms, lid deformities may be corrected by whole-skin or epidermic grafting (Wolfe-Lefort flaps or Thiersch grafts), by the use of pedunculated flaps, and by the Esser epithelial inlay, and the Gillies' epithelial outlay or overlay, although occasionally in very moderate degrees of cicatricial ectropion the V Y operation will suffice.

1. **Wharton Jones' Operation.**—A horn spatula is put into position to protect the eye, and a V-shaped incision is made. The flap is then separated sufficiently to enable the lid to be pushed up into place. The lower part of the wound is drawn together with sutures, thus converting the V into a Y. The triangular flaps should include the cicatrix which has produced the original trouble (Fig. 318).

[1] Plastic operations on the eyelids have necessarily greatly increased in number as the result of war injuries. In many instances the operation is not alone concerned with the eyelids, but owing to extensive or multiple wounds is concerned with the larger problems of maxillofacial surgery, and therefore the plastic surgeon and the ophthalmologist may often find it of advantage to work together. The site and character of the lesion will in each instance determine the best method of procedure, and it would not be possible in a chapter of the present scope to indicate in detail the numerous ingenious methods which have been devised for the correction of cicatricial ectropion by these blepharoplastic operations, or for the formation of an entirely new lid to replace one that has been destroyed by some disease, such as lupus, or by injury. For those interested a large literature, foreign and American, is available. Particular attention is directed to Plastic Surgery of the Face, by H. D. Gillies, Oxford University Press, 1920; Plastic Operations on the Orbital Region, including Restoration of the Eyebrows, Eyelids, and Orbital Cavity; Bowman Lecture by V. Morax, Transactions of the Ophthalmological Society of the United Kingdom, 1919, vol. xxxix; War Injuries of the Eyelids, by John M. Wheeler, Transactions of the American Ophthalmological Society, 1919, vol. xvii, and Dermic Grafts for Correction of Cicatricial Ectropion, Amer. Jour. of Ophthalmology, April, 1920, and Plastic Operations About the Eyes, Trans. Inter. Congress of Ophth., 1922, Washington, D. C.; Reparative Surgery after War Injuries of the Eyes, by G. H. Grout, Archives of Ophthalmology, 1919, vol. xlviii; Plastic Repair of the Eyelids by Pedunculated Skin Grafts, by George H. Cross, Trans. Sec. Ophth. Amer. Med. Assoc., 1921; Handbuch der Ärtzlichen Erfahrungen im Weltkriege, 1922, band v, Augenheilkunde, S. 449.

In cases where the lid deformity is not too elaborate the following procedure may be followed:

2. **Epidermic Grafts for the Correction of Ectropion.**—The ectropionized lower lid, for example, is dissected free of attachments in such a manner that it can be drawn upward well over the upper lid and held in place by three sutures inserted in its margin and passed through a strip of firm zinc plaster fastened horizontally above the brow. All underlying cicatricial bands are divided and scarred tissue removed. The denuded area which presents itself is covered with a large Thiersch graft, which should be cut from the inner aspect of the arm, and which should be larger than the surface it covers, and pressed neatly and smoothly into position. No dressing of any kind need be placed on the graft, but it may be exposed to the air, and even better, when possible, to the sunlight. At first there is considerable wrinkling of the Thiersch graft; gradually this disappears, and ultimately, in successful cases, the surface is smooth. The stitches, if any have been required, are removed at the end of ten days or two weeks.

The tissue of the cheek below the area denuded by the dissection of the ectropionized lid should be supported, to prevent its sagging, by suitable strips of plaster. Naturally, the same procedure applies to an ectropion of the upper lid, which after being freed is drawn downward over the lower lid, and is fastened as before to the tissue of the cheek.

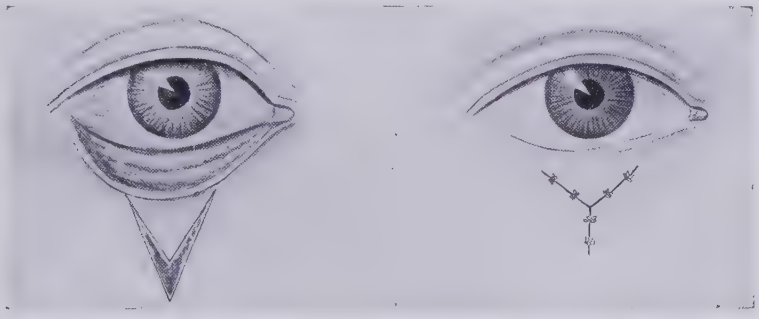


FIG. 318.—Wharton Jones' operation for ectropion.

3. **Epithelial Overlay.**—Should there be decided loss of tissue, as the result of a burn, for example, and an extensive dissection and undermining be required in the removal of scar tissue before the eyelid can be sutured into a favorable position, an *epithelial overlay* (see also page 707), as Gillies suggests, can be utilized as follows:

An impression of the entire raw surface is taken by means of dental modeling composition; next the denuded area is covered with a Thiersch graft and kept firmly in place with the previously prepared mold or impression. This operation, as Hanford McKee points out, may obviate the necessity of forming a pedunculated flap. It is, in fact, an elaboration of the procedure just described.

4. **Free Dermic (Whole Skin) Grafts for Correction of Ectropion.**—In place of a Thiersch (epidermic) graft, as previously described, a free Wolfe (dermic, "whole skin") graft may be employed, which in the opinion of some surgeons, notably John M. Wheeler, far exceeds in effectiveness either an epidermic layer or a pedunculated flap.

The bed which is to receive the graft is prepared practically in the manner already described, by making a skin incision, a few millimeters from the lid margin (usually it is the lower lid), of the same length as the palpebral fissure and thoroughly dissecting out all cicatricial tissue, but sacrificing the muscular tissue as little as is possible. Next the lids are fastened together at three equidistant points (*interrupted tarsorrhaphy* [see page 696]), and the denuded area, all bleeding having been checked without use of sutures, is put on stretch by means of strips of plaster above and below, and a dermic graft prepared as directed on page 712 is transferred into position and carefully sewed in place. The grafted area is greased with White's



FIG. 319.—Cicatricial ectropion of upper lid, following a gasoline burn (from a patient in the University Hospital).

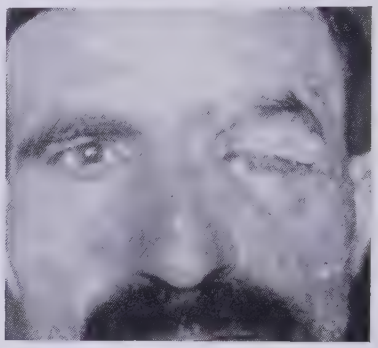


FIG. 320.—Result of operation to correct the deformity shown in Fig. 319, performed according to method described in paragraph 4. The stitches which fastened the lids together have not been removed.



FIG. 321.—Burns of the face and double cicatricial ectropion following a gas burn (from a patient in the University Hospital).



FIG. 322.—Result of operation to correct deformities shown in Fig. 321, performed according to method described in paragraph 4. The stitches which fastened the lids together have not been removed.

ointment or sterilized vaselin (Wheeler) over which a piece of thin rubber tissue is spread; on top of this is placed surgical cotton and the whole secured by means of a pressure bandage. The dressings should not be removed for at least five days.

For simple ectropion Wheeler recommends that, after the bed is prepared as described, the true skin-graft to cover it shall be taken from one of the upper lids. A graft 50 mm. long and 25 mm. wide can be dissected safely from any normal upper lid.

The author agrees with Wheeler and other advocates of this procedure that it represents a most valuable method of dealing with cicatricial ectropion, but he is also convinced that epidermic grafts, as advocated, also have an important relation to the treatment of cicatricial ectropion.

The method of sewing the lids together is indicated if the eyeball is in place; if not, they must be anchored to the orbital contents.

Epidermic Inlay (*Esser's Operation*) **and Epidermic Outlay** (*Gillies' Operation*).—(*a*) Esser's epithelial or epidermic inlay operation was designed originally, in so far as the eyelids are concerned, to correct *entropion*, to enlarge a contracted socket, etc. Its description for the sake of convenience is inserted at this place.

To deepen the lower fornix, for example, the procedure is as follows:

An incision is made from the outside into the lower lid until the deep surface of the conjunctiva is reached. The scar tissue which is found in this situation, due to the injury, is next dissected carefully off the deep surface of the conjunctiva; with some sterilized dental composition a mold is then taken of this cavity. The skin edges are drawn together over the mold in order to ascertain whether it is of the right size. If it is too big, it is cut down until the skin edges meet. The mold is next taken out of the cavity and a freshly cut Thiersch graft from the inner side of the arm is wrapped around the mold, which is put into the cavity, the epithelial surface of the graft being toward the mold. The skin edges are next united over the mold and skin-graft, which should be buried for ten days.

At the end of this time an incision is made through the conjunctiva at the spot where the scar tissue was removed. Immediately underlying the conjunctiva will be found the skin-graft, and next to it the mold. One blade of a pair of scissors is inserted into the cavity in which the mold is lying, and the incision in the conjunctiva thus widened to the full extent of the size of the mold. The mold can be easily removed, and it will be found that the cavity in which it is lying is epithelialized on all its aspects, and the only raw surface to contend with is the small area lying between the skin-graft and conjunctiva. A prosthetic piece should be immediately inserted into the cavity in order that it may retain its size and shape and to keep its opening into the eye socket widely expanded. This is of great importance, and the nearer the inlay is put to the conjunctiva in the first instance, the less raw area there is to heal over. Waldron makes the primary incision in the conjunctiva, and after suitable undercutting buries the graft-covered mold, holding it in place by suturing the conjunctiva over it.

(*b*) Gillies' description of his epithelial outlay, somewhat condensed, follows:

"For ectropion of the upper lid a curved incision is made just above the lid margin. This is deepened slightly, but not down to the tarsal plate, and care is taken to avoid interference with the levator palpebræ superioris muscle. To a certain extent the flap of skin lying above the incision between the incision and the

eyebrow is undercut. This undercutting is continued until the lid margin descends to a lower level than normal.

"A mold is next taken of this cavity, and, covered with an epidermic graft as before described, is put into place, and the skin united again at the original incision. After the skin is thus sewn over the mold the ectropion is more pronounced for the time being and until the mold is removed. This incision is not tightly closed, so that there are one or two gaps in it which allow the new epithelium to grow around the margin of the incision. In some of the cases the incision is reopened and the mold removed on the tenth day. The lid will drop below the normal level, and the ectropion is permanently cured. In other cases the incision is allowed to open gradually of its own accord until there are only one or two small bridges of skin holding it in position. These can easily be cut."

This operation, although successfully utilized by its designer, has not seemed to the author as satisfactory a procedure as the correction of the deformity by means of a free dermic graft.

Pedunculated Flaps.—For the repair of displaced eyelids and loss of lid substance pedunculated flaps taken from the temple and forehead and rotated into place after suitable dissection are constantly utilized, and in the opinion of many surgeons the flap method should generally be the operation of election. The flap may also be cut from the cheek and slid or advanced into position to cover the raw surface which has been exposed by the dissection made necessary to return the displaced lid to its normal relations. Occasionally, a pedunculated flap taken from the cervical region has been employed to restore the lower lid (Snydacker's method).

Naturally, retractions of the temporal portions of the lids are more easily remedied than those of the nasal aspect. But small flaps cut from the forehead and turned down to correct defects near the inner canthus can be utilized, as especially recommended by T. Harrison Butler.

Space does not permit a description of the many ingenious plastic methods devised especially during the recent war. Certain general principles may be mentioned.

The exposed raw surface should be carefully measured with compasses or a template should be made of tinfoil or rubber tissue, to guide the surgeon in marking out the flap which is to be rotated or advanced into position. This flap should be about one-third larger in all directions than the defect it is intended to cover, its pedicle of sufficient size to furnish blood-supply, and it should not be cut too thin. Cicatricial tissue must be entirely removed and cicatricial bands thoroughly divided. Ligation to control hemorrhage should be avoided; during operation pressure and artery clamps should be used to control bleeding, and the vessels twisted before the hemostats are removed. A small gauze drain, to remain a day or two, should be placed at or near the base of the flap. Support of flaps in order to avoid tension may be secured by means of properly placed strips of plaster, and Wheeler employs adhesive strips, equipped with small hooks, so placed that traction necessary to relax the flap will be produced. A rubber band is strung between the hooks to produce tension. Although skin sutures

may be removed in four to five days, this relaxation apparatus should remain in place for at least ten days to assure strong union, with the eyelid in proper position (Fig. 323).

In a certain number of cases a cavity is formed because there has been a depressed adherent flap. This may be filled in with fat or muscle before the flap is sutured into place, and loss of a portion of the orbital margin may be repaired by cartilage grafts.

Should it not be possible to cover in completely the defect on the temple, cheek, or forehead after the flap has been rotated into place, the defect should be covered with a Wolfe or Thiersch graft.



FIG. 323.—Support of flaps as utilized by Dr. John Wheeler (Archives of Ophthalmology, vol. xlix, p. 35).

Should there be breaking down of the epithelium of the flaps, indicating necrosis of the upper layers, the suggestion of Gifford to scrape the area until healthy bleeding tissue is reached, and then apply a Thiersch graft, may be followed with advantage, although the necessity for this is more likely to occur in cases in which a flap without a



FIG. 323*a*.—Pedunculated flap, taken from the temple, showing sutures in place. (Patient in U. S. General Hospital No. 11; courtesy of Captain George Cross.)



FIG. 323*b*.—Pedunculated flap, showing the union soon after removal of the sutures. (Patient in U. S. General Hospital No. 11; courtesy of Captain George Cross.)

pedicle has been transplanted than where flaps retain living connection with the surrounding tissues.

Figures 324 to 330 illustrate a few of the well-known procedures of *blepharoplasty*. Although essentially diagrammatic, they serve a useful purpose in that they show some of the methods by which sliding flaps may be formed for the relief of deformities of the lid. But, as before

noted, successful blepharoplasty usually cannot be secured by means of a named operation, which may well be a guide, but only by a care-

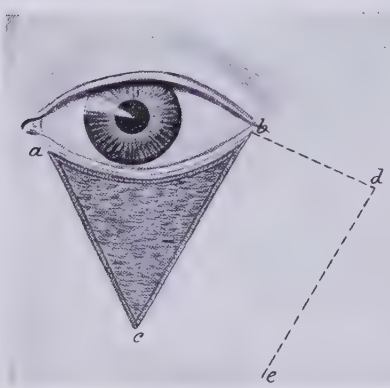


Fig. 324.

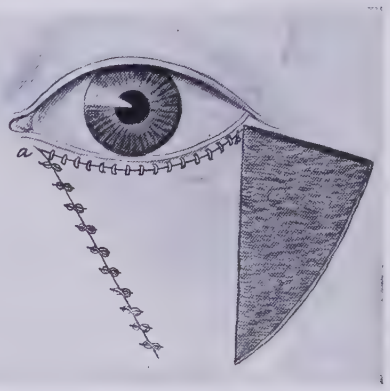


Fig. 325.

Figs. 324, 325.—Restoration of the lower lid by Dieffenbach's method. The diseased or scar tissue has been removed in a triangular flap, *a–b–c*. This defect is covered by a flap taken from the cheek, indicated by the dotted lines, *b–d–e*, with the result shown in Fig. 325. The remaining gap may be covered with a Thiersch graft. This operation has been modified by several surgeons chiefly in the formation of the flap *b–d–e*, so that the secondary defect shall be smaller and the pedicle narrower, allowing an easier rotation, as is the case in the Arlt-Blaskovic modification.

ful plan of action according to the condition of the defect and that of the surrounding tissue. Prior to operation the skin surface should

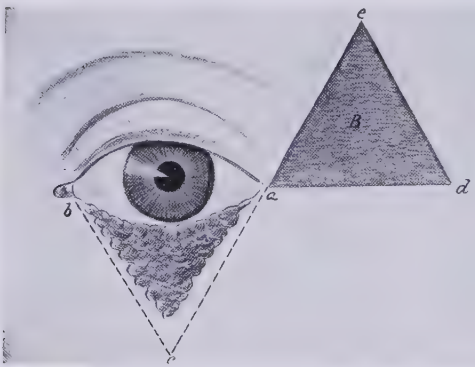


Fig. 326.

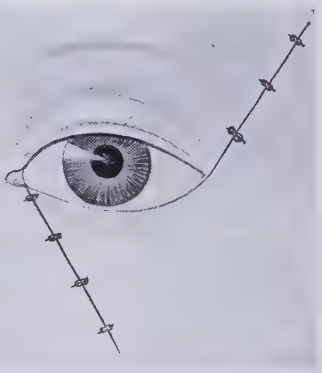


Fig. 327.

Figs. 326, 327.—Restoration of lower lid by Burow's method. The diseased tissue is removed with the flap *a–b–c*. The horizontal incision is prolonged upon the temple and forms the basis of the triangle *a–d–e*. This flap (*B*) being removed, the cutaneous flap *a–c–d* is dissected up and drawn inward so that the angle *a* is sutured at the point *b*, and *a–d* forms the free border of the lid; *c–a* is now united with *c–b*, and *d–e* with *a–e*, with the result shown in Fig. 327.

be thoroughly cleansed with soap and painted in the usual manner with iodin. During the procedure the field of operation should be fre-

quently irrigated with a warm sterile physiologic salt solution. After the flaps are in place they and the suture lines may be again painted with a 5 per cent. solution of iodin; this does not interfere with their vitality (Morax). A dressing of rubber protective on which layers of gauze are laid is not satisfactory, but if the flap is first smeared with

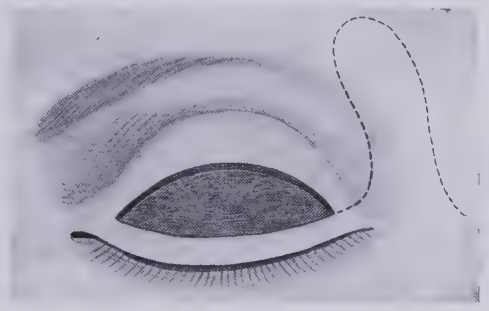


Fig. 328.—Restoration of the upper lid by Fricke's method. The diseased tissue has been removed in an oval flap. The resulting gap is covered by a similarly shaped flap taken from the temple, indicated by the dotted line.

sterile vaselin and then covered with thin rubber tissue on which is placed surgical gauze held with strips of plaster and over this cotton and a firm pressure bandage the results are good (page 707). A cellular tissue with mesh of 1 or 2 mm. impregnated with a mixture of castor oil and wax and sterilized at high pressure, known as "greasy tulle," is

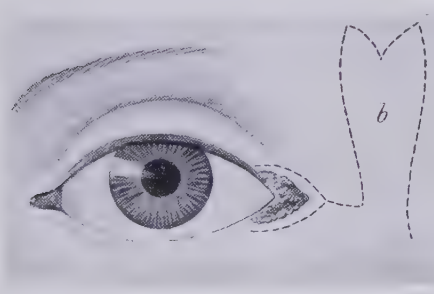


Fig. 329.

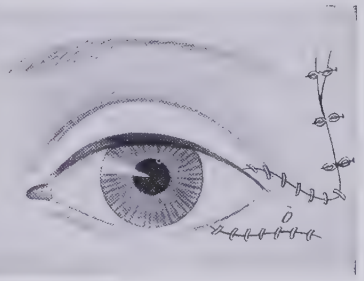


Fig. 330.

Figs. 329, 330.—Restoration of the external angle of the lids by Hasner d'Artha's method. The diseased tissue is removed by two elliptic incisions, and the defect covered with a flap taken from the temporal region at *b*, cut in the manner indicated by the dotted line, with the result shown in Fig. 330. The same operation applies to the inner angle, the flap being taken from the nose.

highly recommended by Morax. At the end of seventy-two hours, should there be any exfoliation of the epithelium, this may be trimmed away and the edge of the flaps anointed with White's ointment (page 762). The dressings suggested may be used also for protecting a whole-skin non-pedunculated flap.

Whether pedunculated flaps or transferred skin-grafts shall be

employed must be decided by the operating surgeon, according to his experience, and this is true as to whether a Thiersch or a Wolfe graft shall be used. The author strongly advocates the various methods of grafting which have been described with which he has had good success, the transference of whole skin-grafts having been especially satisfactory. Naturally, in certain circumstances pedunculated flaps are required. Wheeler recommends them in ectropion only where there has been such loss of tissue that a depression remains to be filled in, consequently a thick flap is required.

Absence of the action of the elevator of the lid and loss of the lid margin of the upper lid present complications which preclude in most instances a satisfactory result; indeed, in these circumstances Gillies considers operation practically useless, that is, operation on the lids and orbital socket, and he doubts if a strip of eyebrow, although it has often been used, constitutes a satisfactory replacement of the eyelashes where the margin of the upper lid has been lost. Wheeler, on the other hand, reports satisfactory results with this operation, taking the graft in the form of a strip preferably from the inner end of the brow and sewing it in place to restore the lost eyelash line, and Morax, in the event of one eyebrow being destroyed and the other brow being thick enough, detaches half its breadth, leaving a nasal pedicle, and fixates the flaps thus secured in an incision on a level with the upper margin of the orbit. Should both eyebrows be lost he performs autoplasty without a pedicle, the flaps being taken from the occipital region.

Operation of Skin-grafting.—If the portion of skin removed for a graft contains only the epidermis, rete Malpighii and the top of the papillary layer, it is known as an *epidermic graft;* if the entire thickness of the skin is included in the graft, that is, the whole skin, it is denominated *dermic graft* (also Lefort-Wolfe graft). A true Thiersch graft should be composed only of the epithelium and the most superficial layer of the cutis.

To remove a whole skin or dermic graft, with a sharp knife the surgeon cuts just through the skin as it has been outlined, slightly undermines the edge of the flap, and proceeds to dissect it from the underlying subcutaneous tissue, which is, therefore, not included in the graft. Having been cleanly removed the graft is transferred to the denuded area. Before completing the dissection fine silk sutures may be passed through the undermined edge of the flap which facilitate, after the dissection is ended, the transfer of the graft to the area to be covered by it; they are utilized to fasten it into proper position. The grafts may be taken from the inner side of the arm or thigh, avoiding hairy areas. Sometimes grafts are secured from other persons (donors) or from an amputated limb possessing sound skin.

To take a *Thiersch graft* the skin, preferably on the inner side of the arm, is stretched flat by an assistant and the surgeon with a long, wide very sharp razor applied flat-wise to the prepared area by means of to-and-fro movements removes the desired tissue which, being very thin, rolls up on the razor from which it may readily be transferred directly to the denuded area. Walter Parker recommends that the skin surface shall be greased with sterilized vaselin, which greatly facilitates the cutting of the graft. The indications for, and the dressings suited to, skin-grafts have been described.

Operations for Prosthesis in Cases of Cicatricial Orbital Sockets.—Owing to the formation of cicatricial bands and scar tissue as the result of burns (acids, alkalies, molten metal), injuries, badly performed enucleations, trachoma, etc., it is often necessary to make a new culdesac before an artificial eye can be adjusted. Numerous operations have been devised to enlarge the socket in these circumstances, mere division of contracting bands being entirely insufficient. It would not be possible in a chapter of such limited scope as the present one to describe the many ingenious and effective procedures which have been elaborated, and therefore only a few general principles and one or two methods can be recorded.

In general terms, for the restoration of an obliterated eye-socket a new culdesac may be formed by pedunculated flaps from skin adjacent to the orbit, or it may be lined by transferred integument (Wolfe grafts), or by Thiersch grafts, molded in place after suitable dissection by means of various conformers. The enlargement of a contracted lower fornix by means of an Esser inlay is elsewhere described (page 707).

Lining a New Culdesac with Epidermic Graft.—A contracted socket, after the new culdesac is formed by suitable dissection and freed from scar tissue, may be lined with a Thiersch graft, wrapped around (the raw surface outward) a mold of dental modeling composition, or "stent." The graft must be somewhat larger than the area to be covered, and the mold of stent so formed that it neatly presses the graft over the entire surface of the new socket. After it is in place the lids should be stitched together, and should not be disturbed for at least ten days. If successful, after removal of the "stent" it will be found that the graft "has taken," and covers the desired surface. Even if a part of the graft is lost, a second grafting over the exposed area is perfectly feasible. In some cases it is possible, before the graft is put in place, to introduce a piece of cartilage, as is described (page 749), which forms a base on which the artificial eye rests.

Weeks restores the culdesac with dermic (Wolfe) grafts which are secured by sutures which anchor them to the periosteum and emerge on the cheek. He insists that success depends on attaching the implanted flaps, whatever their nature may be, to periosteal or epiperiosteal tissue at the margins of the orbit.

Maxwell's Method.—The lower culdesac may be restored by Maxwell's method as follows:

"An incision is made in the floor of the socket and carried downward behind the lower lid. A semilunar flap about 8 mm. in width at its widest part is marked out on the skin, its upper concave border being about 5 mm. below the edge of the lower lid. The incision along the upper border of the flap is made to communicate with the bottom of the wound in the socket. The flap is now dissected up from the subcutaneous tissue, except an area represented by the dotted line in the figure. The two ends of the flap (a' and b') are passed through the opening into the socket and sutured to each end of the socket incision (a and b), and the borders A' and B' being also passed through, are sutured to A and B respectively. The space on the cheek is closed and the operation completed by putting in a temporary glass eye or shell. This should be as nearly as possible of the size and shape as that which is to be ultimately worn. This glass eye prevents the new sulcus from being obliterated by contraction and gives it a suitable shape. It cannot safely be taken out for at least a week, as the skin incision might be opened in so doing. If there be

secretion, the space behind can be flushed out by a lacrimal syringe armed with a fine curved nozzle, which can be introduced under the eye at the inner or outer canthus. A glass shell with a hole in front is preferable to a glass eye, because it allows a syringe to be more easily used, and, being transparent, a view of the part behind can be obtained" (Fig. 331).

For those cases where the socket, although shrunken, retains a certain amount of conjunctiva, Meyer Wiener's method may be used, which consists, after suitable dissection, in providing a conjunctival covering for the lower lid, leaving the bulbar surface to be covered. This is accomplished by wrapping a previously shaped, lead plate with epidermic grafts placed in proper position. To restore the entire orbital socket, Schwenk and Posey form the lower culdesac by Maxwell's method and the upper one by transposing a long flap taken from the skin of the forehead above the brow.

The author has had no experience with Maxwell's method which, however, is commended by some surgeons (Zentmayer), but has also proved disappointing to

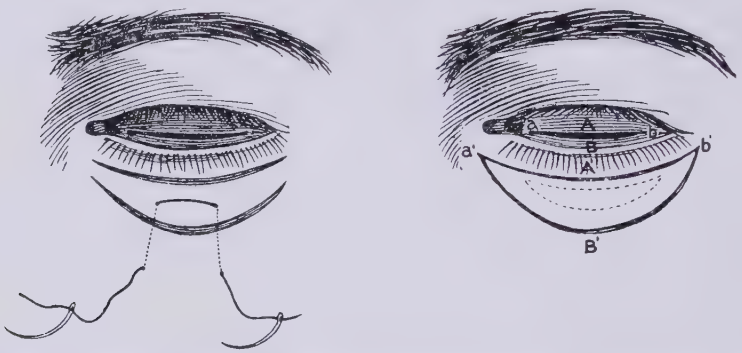


Fig. 331.—Maxwell's operation to enlarge a contracted socket ("Ophthalmic Review," vol. xxii).

others. His best results have been obtained by lining the prepared socket with a mold of "stent" covered with an epidermic graft (page 713).[1]

OPERATIONS ON THE CONJUNCTIVA

Conjunctivoplasty.—The use of conjunctival flaps in the treatment of spreading ulcers of the cornea, after abscission of prolapsed iris, and in the management of corneal and corneoscleral wounds, has been referred to in the sections devoted to these conditions. It represents a surgical procedure of great value and its good results were noteworthy during the past war. The technic is as follows:

If the wound is situated peripherally, for example, at the corneoscleral border, the conjunctiva is incised along one-half of the corresponding circumference of the cornea, undermined sufficiently, and drawn across the cut and fastened by means of two sutures placed at each end of the incision; at least one-half of the cornea can be covered in this manner (Fig. 333). For a wound crossing the cornea almost entirely, or for one situated quite centrally, it is more expedient to cover the defect by means of a bridge of conjunctiva. To form it one cut is directly circumcorneal and a second one placed about 8 mm. from it. The piece of conjunctiva between

[1] For an illustrated description of this technic see an article by John Wheeler, Amer. Jour. Ophth., 1921, vol. iv, 481.

these two incisions is drawn over the injured area and fastened by means of a suture above and below (Fig. 335).

In gaping wounds across the cornea it may be necessary to cover the entire corneal area with conjunctiva. For this purpose the conjunctiva is incised around

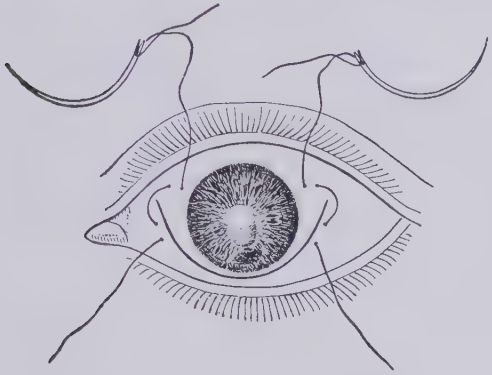


FIG. 332.—By passing the suture through a fold in flap and then through a fold above a firmer hold can be obtained and the anchoring hold should include episcleral tissue. (After Kuhnt, "Medical War Manual," No. 3, Lea & Febiger, 1918.)

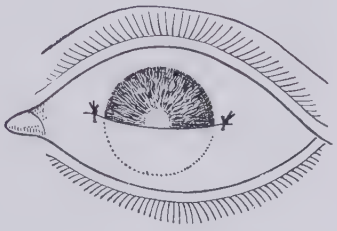


FIG. 333.—Flap in place. (After Kuhnt, "Medical War Manual," No. 3, Lea & Febiger, 1918.)

the entire circumference of the cornea, undermined for approximately 6 mm., and then drawn over the cornea by means of a purse-string suture (de Wecker), or fastened after the manner of Kuhnt (Figs. 332, 335). After the corneal wound has healed the conjunctival covering is removed and restored to its original position.

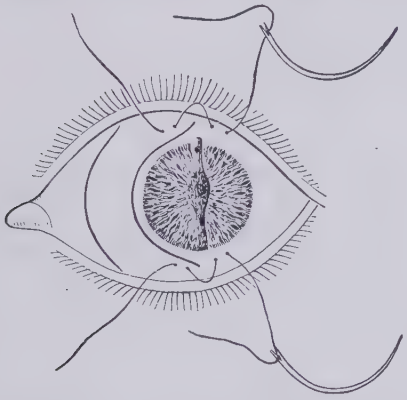


FIG. 334.—Bridge. (After Kuhnt, "Medical War Manual," No. 3, Lea & Febiger, 1918.)

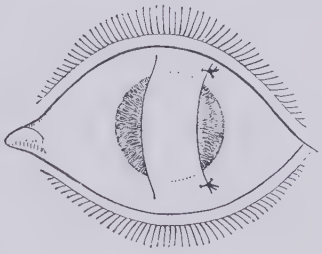


FIG. 335.—Bridge in place. (After Kuhnt "Medical War Manual," No. 3, Lea & Febiger, 1918.)

Even if a wounded eye with prolapse of the iris is not seen until there is incarceration of the iris in the wound, the prolapse, if no infection exists, may be amputated in the usual manner and the opening covered with a conjunctival flap.

In any of these instances should no inflammatory reaction appear (iritis, iridocyclitis) within a few days, the likelihood of its manifestation and the danger of

sympathetic ophthalmia are greatly diminished. If such complications develop in spite of conservative surgery, and the danger of sympathetic ophthalmia arises, radical interference is promptly indicated. Fascia lata grafts have been used as a substitute for conjunctivoplasty, and where a Kuhnt graft has failed of its purpose (I. Whitaker).

Operations for Pterygium.—(*a*) *Excision.*—The pterygium is seized with a toothed forceps, raised from the surface of the eye, and shaved off with a Beer knife from its corneal attachment. It is next turned backward, carefully dissected from the underlying tissues, and excised, together with a triangular piece of conjunctiva. This leaves a somewhat diamond-shaped gaping wound in the conjunctiva, which is drawn together with several sutures. If the conjunctiva overlaps the corneal margin, two small vertical cuts should be made in it at right angles to the line of excision. After the apex of the pterygium has been separated from the cornea, the vascular subconjunctival tissue must be scraped away down to the sclera; otherwise there will be reattachment. The suggestion of Prince to tear loose the pterygium with a strabismus hook instead of separating the point with a knife is a very good one. Complete excision is not applicable to large nor to fleshy pterygia.

(*b*) *Subconjunctival excision* of a pterygium is recommended by S. Lewis Ziegler. The head is removed with Beer's knife, next the body of the pterygium is detached from the sclera with scissors and afterward all hypertrophied subconjunctival tissue is carefully dissected from the conjunctiva; the apex is excised and the remaining flap of pure conjunctiva is dropped back toward the canthus. The wound of the conjunctiva is closed by sutures and tension relieved by small incisions suitably placed.

(*c*) *McReynolds' Operation.*—This operation, which is a modification of Desmarres' method, gives admirable results, and in the majority of cases, so far as the author's experience is concerned, has proved by far the most satisfactory one in this affection. Dr. McReynolds describes his operation in the following words:

"Grasp completely the neck of the pterygium with strong but narrow fixation forceps. Pass a Graefe knife through the constriction and as close to the globe as possible, and then, with the cutting edge turned toward the cornea, smoothly shave off every particle of the growth from the cornea. With the fixation forceps still hold the pterygium, and with slender straight scissors divide the conjunctiva and subconjunctival tissue along the lower margin of the pterygium, commencing at its neck and extending toward the canthus, a distance of $\frac{1}{4}$ to $\frac{1}{2}$ inch. Still hold the pterygium with the forceps, and separate the body of the growth from the sclera with any small, non-cutting instrument. Now separate well from the sclera the conjunctiva lying below the oblique incision made with scissors. Take a black silk thread, armed at each end with small curved needles, and carry both of these needles through the apex of the pterygium from without inward and separated from each other by a sufficient amount of the growth to secure a firm hold. Then carry these cutting needles downward beneath the loosened conjunctiva lying below the oblique incision made by the scissors. The needles, after passing in parallel directions beneath the loosened lower segment of the conjunctiva until they reach the region of the lower fornix, should emerge from beneath the conjunctiva at a distance of about $\frac{1}{8}$ to $\frac{1}{4}$ inch from each other. Next, with the forceps, lift up the loosened lower segment of the conjunctiva and gently exert traction upon the free ends of the threads which have emerged from below, and the pterygium will glide beneath the loosened lower segment of the conjunctiva, and the threads may now be tightened and tied and the surplus portion of the thread cut off, leaving enough to facilitate the removal of the threads after proper union has occurred. It is extremely important that no incision be made along the upper border of the pterygium; otherwise it would gape and would leave a denuded space when downward traction is made upon the pterygium."

The return of a pterygium after excision is not uncommon; occasionally the second growth is thicker than the primary one, and may exceptionally assume a species of keloid formation. After McRey-

nolds' transplantation operation the author has observed no recurrences, nor the formation of cysts. For very thin pterygia, either small or widely spread and not always suited to operation, A. S. Prince recommends weekly applications of *carbon dioxid ice.*

Operations for Symblepharon.—An attempt may be made to remedy this condition by dividing the adhesion and uniting the cut edges of the conjunctiva with sutures, or covering the raw surface left after severing the adhesions with flaps of healthy conjunctiva taken from the unaffected parts of the eyeball (*Teale's operation*), or by dissecting back the symblepharon as far as the retrotarsal fold, doubling it upon itself so as to oppose a mucous surface to the globe, and fixing

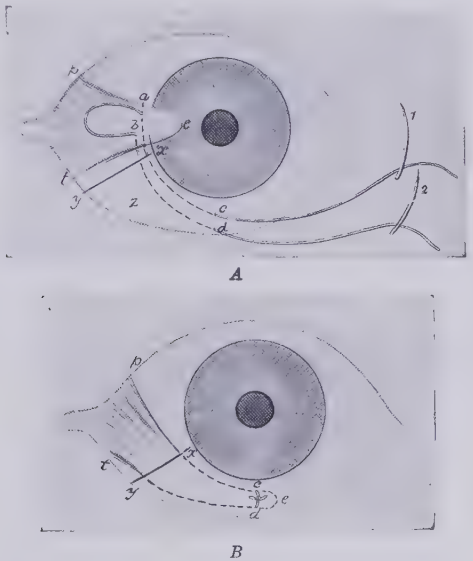


Fig. 336.—McReynolds' operation for pterygium: *A*, showing needles 1 and 2, which enter the neck of the pterygium *p t e* at *a* and *b*, and then pass beneath the loosened lower segment of the conjunctiva *x y z*, and then emerge at *c* and *d* below the cornea; *B*, showing pterygium *p t e* fixed by a single stitch *c d* beneath the loosened lower segment of conjunctiva *x y z*, while the former side of the growth *p x t* is covered by the normal smooth, stretched, and thinned-out conjunctiva *p x t*.

it in this position by means of a ligature which is armed with two needles and passed through the lid from the conjunctiva outward.

Transplantation of Mucous Membrane and of Epidermic or Thiersch's Grafts.—In cases of extensive adhesion between the ball and the lids the transplantation of rabbit's conjunctiva has been attempted.

In this operation, after the adhesions have been severed, the raw surfaces are covered with a flap of conjunctiva taken from a rabbit's eye, so removed as to be free from all submucous tissue, and somewhat larger than the defect which it is expected to cover. It is better to insert the sutures, with which it is afterward put in place, before its removal, as they mark the position of the flap, and at the same time give a means by which it may be transferred from the eye of the rabbit to the eye of the patient. It must be kept warm and moist during the process of transferring it.

All bleeding must be stopped before the attachment is made. Instead of utilizing the conjunctiva from a rabbit's eye, mucous membrane may be taken from the lip or inner surface of the cheek of the patient. In the experience of the author transplantation of mucous membrane is usually an unsatisfactory procedure; but G. B. Jobson prefers mucous to dermic grafts in extensive posterior symblepharon and total symblepharon of the lower lid and has devised a satisfactory technic.[1]

Thiersch grafts may be utilized for this purpose (page 712); they were specially recommended by Hotz, and the author has used them with the greatest satisfaction. The adhesions between the lid and globe are separated in the usual manner, and after all bleeding has stopped the graft is put into position. One difficulty is encountered, namely, the movement of the lid is apt to displace the graft, especially if the denuded area is a large one, and therefore the transplanted skin should be steadied by means of a rigid support best secured by means of a thin mold of dental modeling composition.

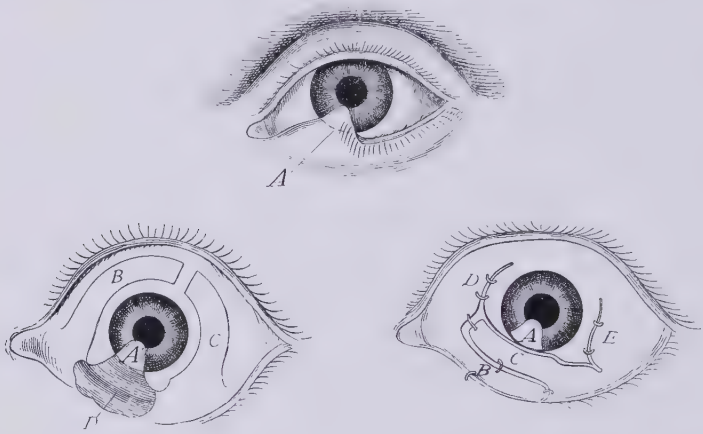


FIG. 337.—Teale's operation for symblepharon (figures from Swanzy). The symblepharon is detached at *A* and removed. Two conjunctival flaps, *B* and *C*, are formed and turned to cover the denuded surface of the eyeball and of the inner side of the lid. The conjunctival gaps are closed by sutures, *D* and *E*.

Operations for Trachoma.—On page 246 the operative procedures suited to cases of trachoma are briefly described. Four methods require more extended notice:

Expression (*H. Knapp's Operation*).—After the patient is etherized the upper lid is everted, seized at the convex border of the tarsus with a pair of fixation forceps, and drawn away from the eye so as to expose thoroughly the whole palpebrobulbar conjunctiva. If the tissue is infiltrated, it may be superficially scarified, preferably with a three-bladed scarifier (Fig. 338). One blade of the roller forceps is pushed deeply between the ocular and palpebral conjunctiva, and the other is applied to the everted surface of the tarsus. The forceps is compressed with some force, drawn forward, and the infiltrated soft substance squeezed out as the cylinders roll over the surfaces of the fold held between it. This maneuver is repeated until all the morbid material has been expressed—in other words, to use Knapp's expression, until the conjunctiva has been thoroughly milked. The lower lid is treated in the same way. During the operation the surfaces should be frequently flooded with a tepid solution of bichlorid of mercury, 1 : 8000, and after the operation cold compresses may be laid on the lid for twenty-four hours.

[1] Trans. Amer. Academy of Ophthalmology and Otolaryngology, 1919, p. 166.

The following day the lids should be everted, and usually a delicate grayish layer of lymph will be found covering the entire area of operation. This should be removed, the swollen mucous membrane exposed, and touched in the ordinary way with a solution of nitrate of silver, 5 to 10 grains (0.324–0.650 gm.) to the ounce (30 c.c.). Each day this treatment should be repeated until the swelling has subsided, when the daily application of a crystal of sulphate of copper is advisable.



Fig. 338.—Three-bladed scarifier.

The operation should be done thoroughly, care being taken to include the commissural portions of the conjunctiva, and the subsequent local treatment of the case must not be neglected. Expression is especially valuable in cases of spawn-like granulations (follicular trachoma) and diffuse hyaline infiltration. It may be used in cicatricial trachoma where patches of hyaline degeneration are present. If the patient suffers a relapse, as he may, the operation should be repeated. In a somewhat extended experience the author has never seen

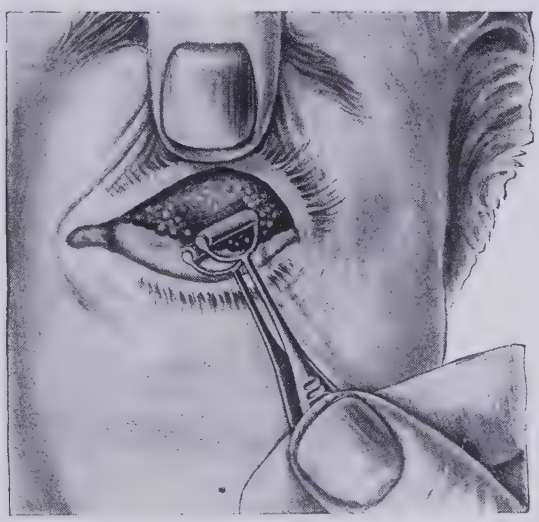


Fig. 339.—Knapp's operation for trachoma (Hansell and Sweet).

any save good results from this method of treating trachoma. It should never be used in so-called acute trachoma. Some surgeons consider the operation more effective if after the expression a germicide is brushed into the tissues.

Combined Expression.—This procedure, and certain modifications of it, is much employed. It is especially recommended by Harvery J. Howard as the result of his large experience with trachoma in China.

The "follicles" having been picked open with a cataract knife, their contents are expressed with Prince's forceps, or squeezed out by means of pressure exerted

with the aid of gauze or cotton sponges wrung out of a saturated boric acid solution, or a 1 : 5000 bichlorid solution. Instillation of a few drops of olive or of paraffin oil completes this stage of the procedure. The patient is required to return on the following day, when the everted lids are gently rubbed with swabs dipped in oil, whereby any adhesions which may have been formed are broken up. Depending upon the severity of the case, after cocain anesthesia, "rubs" with a cotton wound applicator, dipped first in bichlorid solution (1 : 1000) and next in dry boric acid powder, are repeated daily, or every other day, followed by iced compresses. When six such treatments have been given, the strength of the bichlorid solution is increased to 1 : 500. In general terms, discomfort usually ceases after the first three or four treatments. If necessary, and to shorten the length of treatment, a second expression operation may be, and often is, performed.

Modified Brossage.—In place of the operation just described the technic advised by Surgeon John McMullen of the United States Public Health Service may be adopted:

"The eyelid is everted by means of a special forceps. Next by the use of two scalpels, one in each hand, the conjunctiva is gradually raised and the full extent of the culdesac is exposed and the granulations are scarified superficially, beginning from the bottom and extending forward toward the ciliary margin. Succeeding this, in some cases, it is well to use a moderately stiff brush with bichlorid solution 1 : 2000. The next step is to use fine mesh gauze sponges, and these are rubbed over the entire affected conjunctiva until the surface is smooth and the hypertrophy and granulations have been removed. This can be determined by the reappearance of the small blood-vessels to view. The operation is completed by again everting the eyelid and thoroughly washing all blood-clots out of the conjunctiva, etc., with a boric acid solution, followed by the instillation of 2 drops of a 20 per cent. solution of argyrol. The after-treatment consists in cleansing the eyes every three hours with a boric solution and the instillation of a 20 per cent. argyrol solution. This is continued for several days, or until all sloughs have disappeared.

"If a radical operation has been performed the eyes should be examined carefully for the next twenty-four to forty-eight hours for adhesions, and these should be broken up immediately. At the end of about one week following operation, if granulations or rough surfaces are found, these should be lightly touched with a 2 per cent. solution of silver nitrate, repeated two, three, four, or more times a week."

Grattage with the aid of a tooth brush, carrying a solution of bichlorid of mercury, that is, scrubbing the affected conjunctiva, is an operation which does not appeal to the author.

Simple Excision of the Retrotarsal Fold.—A subconjunctival injection of cocain (1 per cent.) causes the diseased transition fold of the conjunctiva to bulge forward, and makes plain a line of demarcation between the diseased area and the healthy bulbar conjunctiva. The convex margin of the tarsus is brought within the grasp of two pairs of forceps, and an incision is made in the healthy scleral conjunctiva close to the line of demarcation from the outer to the inner canthus. Müller's muscle, which has a bluish look, is usually recognized when the wound separates and the bulbar conjunctiva retracts. Next, three sutures are introduced through the margin of the bulbar conjunctiva, which is undermined. The next incision is so placed as to separate the transitional fold from the tarsus; the diseased tissue lying between these two incisions, being seized at the inner canthus, is separated from the underlying tissue with blunt scissors and removed. Finally, the needles attached to the sutures already in place are placed through the edge of the tarsus and tied.

Should the lower fornix be selected for this operation, the upper lid is held back and the patient required to look upward. Next, the surgeon everts the lower lid and excises the required strip of conjunctiva. Sutures are rarely necessary. In both instances following the operation the eyes should be freely irrigated with a saturated boric acid solution or one of bichlorid of mercury (1 : 8000), and the operated area dusted with finely powdered iodoform, following which a light compressing bandage should be applied.

This operation is suitable if the trachomatous process is largely confined to the transition fold, and there is no serious lymphoid infiltration of the tarsus itself. It is also sometimes effective in checking a developing pannus.

Combined Excision.—After free cocainization, the eye being rotated downward, the upper lid is doubly everted and held in position by means of two fixation forceps, in such a manner that the bulbar conjunctiva is drawn upward upon the surface of the tarsus. The first incision, which should penetrate the conjunctiva alone, is made transversely at the juncture of the palpebral and bulbar conjunctiva

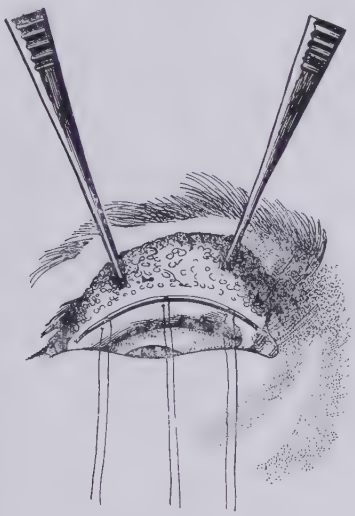


FIG. 340.—Combined excision in trachoma: First stage. (Wootton, Archives of Ophthalmology, vol. xxxix, p. 110.)

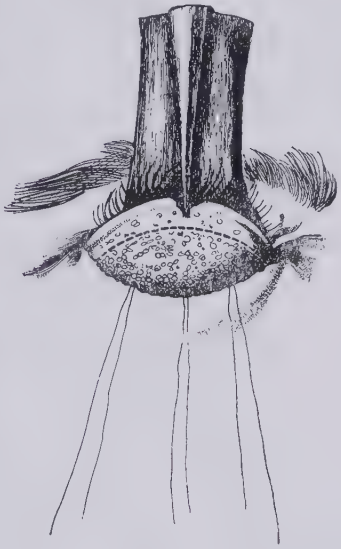


FIG. 341.—Combined excision in trachoma: Second stage. (Wootton, Archives of Ophthalmology, vol. xxxix, p. 110.)

(Fig. 340), thus separating the diseased and healthy tissue. Injury of Müller's muscle, which lies directly beneath, must be avoided. The retracted bulbar conjunctiva is next separated from the subjacent tissue for a distance of 4 mm. Three sutures armed with a needle at each end are inserted through the lower lip of the wound. Following this dissection the lid is allowed to take the position of single eversion, and a horn shield or Jaeger plate is placed beneath the cutaneous surface of the eyelid, the margin of which is pressed firmly upon it (Fig. 341). Next an incision is made for the entire length of the lid 2.5 mm. from its inner margin and exactly parallel to it. The lateral horns of the two incisions are joined by a short vertical cut at their external and internal extremities. Thus the boundaries of the diseased conjunctiva and tarsus are fixed. The next step consists in dissecting up this area, care being taken not to injure the orbicularis or Müller's muscle. How much of the diseased tarsus shall be removed depends upon the severity of the condition and the distribution of the lesions; usually the piece removed is about 2.5 cm. long and 1 cm. broad. Hemorrhage having been checked, the operation is completed by stitching the margin of the bulbar conjunctiva to the rim of tarsus which

remains, and it is important that the conjunctiva shall be united exactly to corresponding points of the tarsus. The eye is closed and the surgeon makes gentle traction on the middle suture in a direction vertical to the lid margin. The point where the suture crosses the upper margin of the tarsal rim is grasped with toothed forceps, one blade being passed beneath the lid, which is then everted. The suture is next passed through the upper margin of the tarsal cartilage at the point designated by the teeth of the forceps. The other sutures are treated in like manner (Fig. 342). In order to avoid pressure on the cornea the sutures may be placed thus, following the method of von Blacowicz. The sutures are armed with two needles, which are passed entirely through the lid, the anterior one transfixing the upper margin of the cartilage, the posterior one the aponeurosis, muscle and skin in close proximity. The sutures are tied over a roll of gauze, and may be removed on the fifth day.



Fig. 342.—Combined excision in trachoma: Placing the sutures. (Wootton, Archives of Ophthalmology, vol. xxxix, p. 110.)

This mucotarsal excision is recommended for chronic trachoma with tarsal infiltration, chronic trachoma with pannus independently of the tarsal condition, and in gelatinous trachoma of the retrotarsal folds and thickening of the tarsus.

In case of chronic trachoma, associated with great infiltration and thickening of the tarsus, Kuhnt recommends *extirpation of the tarsus* with excision of the conjunctiva.

Subconjunctival Injections.—The indications for these injections have been given in connection with the diseases for the relief of which they have been recommended, and, in the experience of the author, they are sometimes useful, particularly in various inflammations of the uveal tract, the sclera, and some types of parenchymatous, as well as ulcerated, keratitis. Their value in detachment of the retina, especially solutions of salt, has been described (see page 516). It should be remembered that repeated injections of this character may cause a chronic hyperemia of the conjunctiva and induration and hypertrophy of the subconjunctival tissue.

The eye is thoroughly cleansed and anesthetized by the instillation of a 4 per cent. solution of cocain. The patient is required to look strongly downward and inward in order to expose the supero-external portion of the eyeball. Next, the needle of a hypodermic or Pravaz syringe, properly sterilized and charged with the fluid, is introduced very much in the same manner as when an ordinary hypodermic injection is given, well beneath the conjunctiva and away from the cornea. The quantity to be injected depends upon the nature of the case and the character of the fluid employed. If, for example, bichlorid of mercury is used in a strength of 1 : 1000, each division of a Pravaz syringe would contain $\frac{1}{20}$ mg. of the drug. Ordinarily, a solution of bichlorid of mercury, 1 : 2000 or 4000, may be used, and from 4 to 8 minims (0.24–0.50 c.c.) injected. Generally, cyanid of mercury is the preferable drug, and may be used in a strength of 1 : 2000 to 5000. The injection may be rendered practically painless by adding a few drops of a 1 per cent. acoin solution to the fluid. Darier's directions are to add one-third of a syringeful of a 1 per cent.

solution of acoin to two-thirds of a syringeful of cyanid of mercury, 1 : 1000, and, therefore, obtain a solution of 1 : 1500. Physiologic salt solution is efficient and much less painful. From 15 to 25 minims (0.92–1.54 c.c.) may be injected. Solutions of hetol (cynamic acid) in 1 per cent. strength have been advised by Pflüger. Subconjunctival injections of guaiacol cacodylate are recommended in tuberculous sclerokeratitis and uveitis (Török, Darier).

OPERATIONS ON THE CORNEA

Paracentesis Corneæ.—The local application of cocain is usually sufficient, but in nervous subjects and young children general anesthesia may be necessary. The operation is performed as follows:

The cornea is punctured near its lower margin or, in the case of an ulcer, through its floor with a paracentesis needle constructed with a shoulder to prevent an undue depth of entrance, and inserted at an angle of 45° with the point of contact; or with a broad needle held flatwise, the point being kept well forward so as to avoid wounding the lens. By rotating the needle slightly on its long axis the lips of the opening are separated and the contents of the aqueous chamber more readily escape. The needle must be withdrawn slowly, lest a sudden gush of aqueous cause prolapse of the iris. The eyeball may be steadied by a spring speculum (see Fig. 345) or fixation forceps (see Fig. 346), provided the former does not put too much pressure on the globe, or the lids may be separated by the surgeon's fingers. If it is necessary to reopen the wound, the probe end of the instrument should be used.



Fig. 343.—Paracentesis needle.

Application of the Actual Cautery.—The indications for this application in corneal disease are given on page 280. If possible, a suitable galvanocautery should be employed. If this is not at hand, a platinum probe fixed in a handle similar to the one which is attached to a laryngoscope mirror will suffice. The operation is done as follows:

A few drops of procain or butyn solution are instilled to produce anesthesia, and the probe or the point of the cautery is brought to a red heat, transferred to the area of disease, and all the sloughing material, and particularly the edge of the ulcer, is gently but thoroughly cauterized. It is not necessary to burn beyond the edge of the ulcer into sound tissue. The extent of the ulcerated area, even to the finest spot characterized by loss of epithelium, may be ascertained by the use of fluorescein, but it should be remembered that this drug also colors, but less vividly, diseased epithelium, and hence is apt to stain the epithelium for some distance surrounding the ulcer. Ulcers with much necrotic tissue on them stain yellow. The separation of the lids with a stop speculum is needless; in fact, this is disadvantageous on account of the pressure it exerts upon the eyeball. They may be parted by the hands of the operator himself. After the operation the eye should be washed out with boric acid solution, a drop of atropin instilled, and a bandage applied.

Guthrie-Saemisch Section.—The upper lid being raised on an elevator by an assistant, the surgeon proceeds as follows:

The conjunctiva below the cornea is seized with fixation forceps, a cataract knife is entered on one side of the cornea, carried across the anterior chamber to the other side of the ulcer, and the section made with its cutting-edge forward, directly through the diseased area, evacuating the collection of pus in the layers of the cornea and at the bottom of the anterior chamber. If the hypopyon is tenacious, this may be removed by inserting a delicate pair of forceps through the in-

cision and seizing the slough, or it may be washed out with a specially devised syringe. If the pus reaccumulates, the wound should be reopened with a probe and the contents of the anterior chamber again evacuated. Schwenk has modified this operation in that after puncture and counterpuncture are made he presses the knife backward to equalize the tension and by rotating it on its long axis creates a gap at its point of entrance and exit through which the fluids drain and gradually release the tension; finally, the knife edge being turned forward, the incision is slowly completed in the ordinary manner.

A great objection to this operation is the danger of prolapse of the iris, which, however, is said to be prevented by Schwenk's technic; indeed, owing to the improvement in the treatment of hypopyon-keratitis by means of various local measures, by thermotherapy, bacterins and foreign proteins (see page 282) the operation is much less rarely performed than in former times. As a rare complication intraocular hemorrhage has been reported (A. W. Sichel).

Operations for Staphyloma.—If the measures used to prevent the formation of staphyloma have been unsuccessful (see pages 290 and 291), an operation must be done for its relief. In partial staphyloma vision may sometimes be improved by iridectomy, and even by a double excision of the iris, but very often these measures fail, and then its removal may be necessary.

A useful operation for the reduction of the size of a partial staphyloma is recommended by Sir George Berry:

A cataract needle is introduced through the base of the staphyloma and held in one hand. An elliptic piece of the cicatricial tissue of which the staphyloma is composed is next cut out by making one incision at one side of the needle with a cataract knife, and another from the other side, converging toward the first, and in such a manner that the portion held by the needle, and consequently the needle itself, is cut out. The dressing consists of a firmly applied antiseptic bandage, and usually it is necessary to continue the bandage for some time until flattening of the mass has been secured.

After excision of a small staphyloma it is sometimes possible to promote healing by uniting the resulting wound margins with fine silk sutures or better by conjunctivoplasty (page 714).

S. Lewis Ziegler's Trefoil or Stellate Keratectomy for the Relief of Anterior Staphyloma.—It may be performed with a punch, thus: A vertical incision is made through the base of the staphyloma, and the inferior blade of a punch is passed into the anterior chamber and beneath the cornea. The punch is closed and the left lateral flap is excised, and in similar manner the right lateral flap is removed. Next the upper margin of the oval wound is grasped at its center by the punch and a vertical piece excised. The trefoil opening is closed with sutures.

De Wecker's Method.—This is suited to complete staphyloma limited to the cornea.

Four sutures should be inserted in the conjunctiva after it has first been carefully detached from the corneal margin almost as far as the equator of the eye. In order to avoid confusion at the moment of tightening the threads, the precaution should be taken of having them of different colors. The removal of the staphyloma is performed by transfixing it through the middle and cutting outward, then seizing the end of the flap thus formed, and removing the rest with scissors. Care must be taken that the lens escapes from the eye. When this is ascertained, the sutures in the conjunctiva are tightened and the conjunctiva drawn over the wound.

In most instances of complete staphyloma, with participation of the sclera, the best operation is enucleation or one of its substitutes.

Tattooing the Cornea.—In order to conceal the disfigurement of a dense leukoma it has been suggested to tattoo the white tissue. This is done as follows:

India-ink rubbed up with water into a fine paste is placed close at hand. After the cornea is cocainized, the eye is steadied with the fingers, and a drop of the pigment is applied to the surface of the leukoma, and the ink pricked into place with the needles. These needles may be fixed exactly at the same level, precisely as if they were all fastened into a small circular piece of cork, or they may be placed side by side (Fig. 344). Finally, a single needle, somewhat of the type of an ordinary cataract needle, may be employed, and the pigment pricked into the tissue with little stabs made in an oblique direction. According to the late Dr. Noyes, the pigment should be prepared by allowing the India-ink stick to soak for several hours in water until it becomes of the consistence of thick paste. A piece of paste equal to the size of the spot to be colored is then placed upon the leukomatous area and pricked into position with the needles. The tattooing should proceed until a uniform black surface is secured. The excess of pigment can be washed away with a saturated



FIG. 344.—Tattooing needle.

solution of boric acid. It has been suggested by some surgeons to use variously colored pigments in order to attempt to reproduce the colors of the iris, and an elaborate technic in this respect has been developed by S. Lewis Ziegler.

Operations for Conical Cornea.—Of the various procedures, briefly summarized on page 312, cauterization of the apex of the cone (sometimes in a triangular manner, the apex of the triangle corresponding to the apex of the cone [Posey]) by means of galvanocautery (the small round extension point designed by H. Knapp is particularly useful) furnish the best results; as the resulting scar may overlie the pupil an optical iridectomy may be required; tattooing the contraction-scar has been advocated. But the visual results are by no means always encouraging; the cauterization may require repetition; it has been followed by glaucoma, cataract, and iritis. Other operations have been tried. Thus the cauterization may be preceded by a corneoscleral trephining, which reducing the tension prevents later bulging of the scar. A. S. Green and L. D. Green operate by a method which is "virtually a combination of the Elliot and La Grange operations." Recently special operative procedures have been devised by Meyer Wiener and by L. Webster Fox. For details of these operations the original descriptions should be consulted.[1]

The methods of *removing foreign bodies* embedded in the cornea and the management of corneal wounds have been described (see pages 313, 315, 714).

[1] A. S. and L. D. Green, Amer. Jour. Oph., June, 1920; Meyer Wiener, Jour. Amer. Med. Assoc., Sept. 8, 1917; L. W. Fox, Trans. Ophth. Soc. of A. M. A., 1919.

OPERATIONS UPON THE IRIS

Iridectomy.—The following instruments are necessary: A stop speculum, fixation forceps, bent keratome, narrow Graefe knife, iris forceps, blunt hook, iris scissors, and horn or metal spatula. The preparation of the patient is described on pages 684, 761, 762. The operation is performed thus:

The patient being in a recumbent position and the eye being under the influence of cocain (procain or butyn is preferred by some operators), unless the case is one of

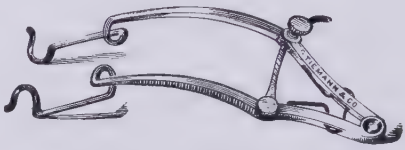


Fig. 345.—Eye speculum.



Fig. 346.—Fixating forceps.



Fig. 347.—Keratome.



Fig. 348.—Curved iris forceps.

acute glaucoma, where a general anesthetic is generally preferable, the surgeon separates the lids by means of a speculum, fixates the eye by seizing with forceps the conjunctiva and subconjunctival tissue at a point directly opposite to that of the proposed section, and introduces the lance-shaped keratome in the following manner: The point of the knife is brought into contact with the apparent corneoscleral margin, or, in some instances, about 1 mm. from the junction of the sclera with the cornea, and in a direction at right angles to the cornea, which direction it keeps until the point just penetrates the anterior chamber. The handle is then well depressed, so that the point of the knife shall not wound the iris or lens, while the blade is slowly thrust onward, until the section is of the desired extent (see Fig. 352). The knife is next slowly and cautiously withdrawn, with its point well forward toward the posterior surface of the cornea, so as to allow a slow escape of the aqueous humor and to avoid scratching the capsule of the lens.



Fig. 349.—Blunt hook.

The *first stage* being completed, the fixation forceps is handed to an assistant, who rotates the globe a little downward, if the section has been made upward, and the surgeon introduces the curved iris forceps, expanding the blades so as to grasp the iris at least 2 millimeters from the pupillary margin, cautiously withdrawing the forceps with the included portion of the iris, and snipping off the latter close to

the wound with a delicate pair of curved scissors (Fig. 350). The scissors may be applied in the manner shown in the figure, or they may approach the withdrawn iris



Fig. 351.—Spatula and probe.

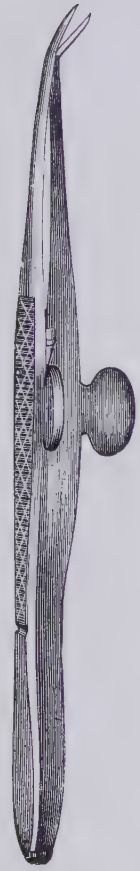


Fig. 350.—Iris scissors.

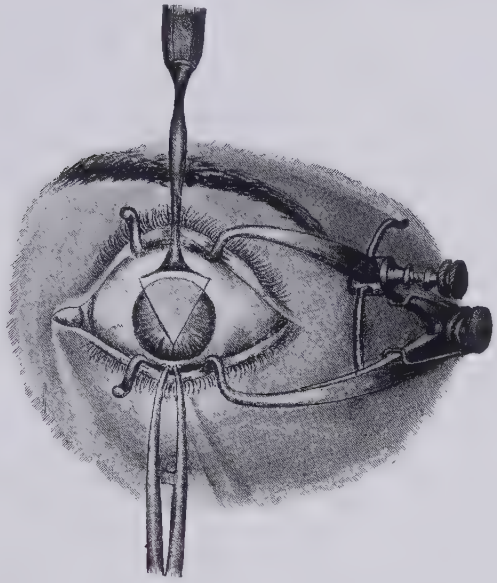


Fig. 352.—Operation of iridectomy: Keratome within the anterior chamber.

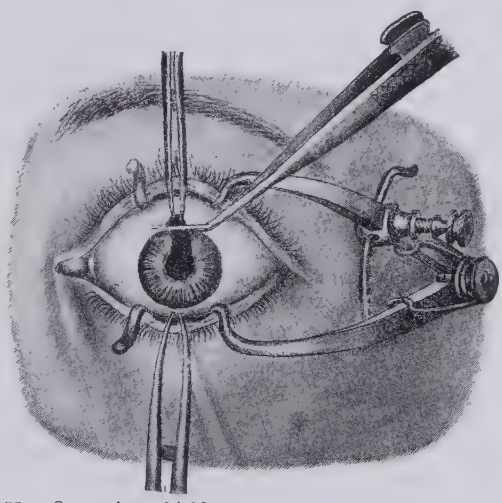


Fig. 353.—Operation of iridectomy: Excision of the piece of iris.

from below, the expanded blades being passed over the cornea until they include the iris tissue, which is then abscised (see Fig. 353).

If the anterior chamber is shallow, it is safer to substitute for a keratome a Graefe cataract knife, making a puncture and counterpuncture, and then cutting in the same manner as the corneal section in cataract is made. Many surgeons prefer this method of making the section in all iridectomies (see page 764).

If the section of the iris should cause hemorrhage into the anterior chamber, an attempt may be made to remove the blood by separating the lips of the wound with a metal spatula (Fig. 351) and making very cautious pressure on the cornea, but triturating movements carried on to any great extent are done at the risk of bruising the lens and causing cataract. The conjunctival culdesac should be irrigated with a warm physiologic salt solution, and the length of the wound, and especially its angles, should be inspected to ascertain if the iris is entangled. Should there be any entanglement of the iris, this must be carefully disengaged with the spatula or olive-pointed probe until the angles of the wound are entirely clear of iris tissue and the pillars of the coloboma perfectly in place. If the wound appears clear, the eye is dressed in the same manner as after cataract extraction (see page 767). One or both eyes may be bandaged, or covered with a pad of aseptic gauze held in place by strips of adhesive plaster. The author prefers to bandage both of them for the first forty-eight hours. Almost always the healing is kind, the anterior chamber is quickly restored, and the bandage may be removed at the end of forty-eight hours and the patient directed to wear a shade or dark glass.

This, in general terms, describes the method of performing an iridectomy, which, however, may require certain modifications according to the indications and according to the judgment of the operator.

1. *Position of the Operator.*—The operator may stand behind the patient's head and push the knife from him if he is making an upward section, or he may stand in front of the patient and push the knife toward him in a similarly made section. The latter procedure has been recommended if the anterior chamber is shallow, as the operator can more readily watch the point of the knife. This direction refers to the lance-shaped keratome. The author prefers to stand behind the patient's head.

2. *Point of Entrance of the Keratome.*—This depends upon whether the iridectomy is for optical purposes or for the relief of increased intraocular tension. If for the former, its position should be exactly at the apparent corneoscleral border; if for the latter, farther back, about 2 mm. from this position, passing through the sclera. Should a cataract knife be used, preferable always if the anterior chamber is shallow, the position of the operator and the manipulations are the same as in the extraction of cataract (see page 764).

3. *Position of the Iridectomy.*—If the iridectomy is for optical purposes, the point of selection is governed by the condition of the cornea. The best position for an artificial pupil is inward or inward and downward, other things being equal. In optical iridectomy good results are obtained and pain lessened by drawing out that portion of iris which is to be excised with a small blunt hook. In place of *optical iridectomy*, as described, Axenfeld performs *precorneal iridotomy*, in which the iris is made to prolapse through a small corneal incision and is incised with a radial scissor-cut, not excised, and carefully replaced. A gradual separation of the cut takes place.

If the operation performed is to restore a pupil to an iris which has been bound down by extensive synechiæ, that portion of the iris should

be excised which is least attached. Generally it is best to perform the section upward and make a broad iridectomy. The same is true if the operation is performed for a partial cataract, although its exact position must be governed by the condition of the lens.



Fig. 354.—Healed corneal section after iridectomy (from a specimen prepared by Dr. C. M. Hosmer in the author's laboratory).

4. *The Width and Depth of the Coloboma.*—Figures 355–357 portray three forms of iridectomy: namely, a broad peripheral iridectomy, as in glaucoma; a small iridectomy, with preservation of the ciliary border; and a narrow iridectomy, for instance, for optical purposes. As noted on page 728, if the iridectomy is performed for the release of increased intra-ocular pressure the point of entrance of the keratome must be in the sclera (2–4 mm. back of the corneal margin) in order to secure a peripheral abscission of the iris which may be obtained by gently drawing the seized iris from one side of the incision to the other before it is cut off.

R. G. Reese has developed a technic whereby the iridectomy, especially for glaucoma, is done under a conjunctival flap. A special broad keratome is used and its point made to engage the conjunctiva 7 mm. from the limbus, and is pushed forward to within 2 mm. of the corneal margin, where the sclera is penetrated obliquely, and the point of the instrument enters the chamber anterior to the plane of the iris, which is seized with forceps and drawn backward and to the left and forward and to the right in order to tear loose the ciliary attachment and abscise it.[1]



Fig. 355. Fig. 356. Fig. 357.

Fig. 355.—Broad peripheral iridectomy.
Fig. 356.—Small iridectomy with ciliary border preserved.
Fig. 357.—Narrow iridectomy for optical purposes. (Modified from Swanzy.)

Complications.—These may occur during the operation or after its completion, and include entrance of the point of the keratome between the lamellar of the cornea, due to beginning the section with the instrument held obliquely and recognized usually by failure of any aqueous humor to escape; touching the iris with, or entangling it upon, the point of the keratome or knife; prolapse of the iris into the wound during the escape of the aqueous, generally of no consequence and, if anything, facilitating its abscission; injury to the lens either with the iris forceps or because the point of the keratome touches and pricks the



Fig. 358.—De Wecker's pince-ciseaux.

capsule—a serious accident, as cataract is sure to supervene. Occasionally a sudden movement on the part of the patient when the iris is grasped with the forceps causes an *iridodialysis* and troublesome hemorrhage. In iridectomies where the iris has been inflamed or is partly atrophic, the tissue being very friable, it may be difficult to seize it accurately, or it comes away in fragments; in these circumstances a blunt iris hook is of advantage. Slow closure of the wound is rare except after iridectomy for acute glaucoma, where usually

[1] For a full illustrated description of the technic of the operation see Trans. Sec. of Ophth. A. M. A., 1923.

it is not disadvantageous. Postoperative infection is exceedingly uncommon; occasionally, in glaucoma, intra-ocular hemorrhage with extrusion of vitreous has occurred.

Iridotomy.—This operation, which is designed to manufacture an artificial pupil, is commonly selected for eyes in which the lens is absent, as after cataract extraction, and in which the pupil has become entirely occluded on account of iridocyclitis. It may be performed by simply splitting the fibers of the iris with a knife-needle, the retraction usually affording a sufficient pupil; or a blunt hook (see Fig. 349) may be introduced and the operation converted into a small iridectomy; or a triangular-shaped piece of the iris may be excised with delicate scissors introduced through a corneal wound (*iritoëctomy*). The operation of de Wecker is performed as follows:

A small triangular keratome, preferably fitted with a shoulder, is entered into the apparent corneoscleral margin and pushed on until an incision of about 5 mm. is made. It is then slightly withdrawn and again inserted, this time causing the point to pierce the iris or the membrane which it is desired to divide. The instrument is now withdrawn, and the delicate forceps scissors of de Wecker is introduced as follows: The instrument is inserted flatwise with closed blades through the wound.

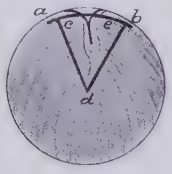


FIG. 359.—Iritoëctomy (one method): *a–b*, Cornea-iris incision; *a–b*, *b–d*, excision of iris membranes.

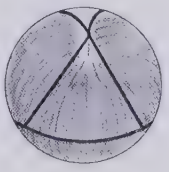


FIG. 360.—Iritoëctomy—another procedure (after Czermak).

One blade is made to pass through the opening in the iris or membrane and the other in front of it. The blades are now pushed onward as far as necessary, closed after the manner of a pair of scissors, and withdrawn. The cut thus being made across the line in which there is the greatest tension, retraction takes place, and if the operation is successful, a useful pupil results (*simple iridotomy*). Instead of this procedure, after the narrow keratome which has pierced the cornea and made a small (2 mm.) opening in the iris-membrane is withdrawn, the iris scissors may be introduced, as before described, and two oblique cuts may be made from either extremity of the incision toward the apex of a triangle, forming thus a triangular flap which is removed with forceps (*iritoëctomy*).

A more satisfactory operation than de Wecker's iridotomy or iritoëctomy is one devised by S. Lewis Ziegler, with which procedure the author has had the most gratifying results and which he highly commends.

V-shaped Iridotomy (*Ziegler's Operation*).—The instruments required are a speculum, fixation forceps, and Ziegler's modified Hays' knife-needle. It is performed as follows:

First Stage.—With the blade turned on the flat, the knife-needle is entered at the corneoscleral junction, or through the upper part of the cornea (Fig. 361), and

passed completely across the anterior chamber to within 3 mm. of the apparent iris periphery. The knife is then turned edge downward, and carried 3 mm. to the left of the vertical plane (Fig. 362).

Second Stage.—The point is now allowed to rest on the iris-membrane, and with a dart-like thrust the membrane is pierced. Then, without making pressure on the tissue to be cut, the knife is drawn gently up and down with a saw-like motion, until the incision has been carried through the iris tissue from the point of the membrane puncture to just beneath the point of the corneal puncture. This movement is made wholly in a line with the axis of the knife, the shank passing to and fro through

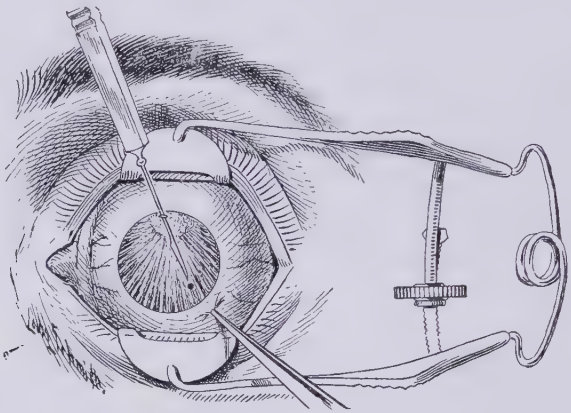


FIG. 361.—Ziegler's V-shaped iridotomy: Knife-needle entered through cornea.

the corneal puncture, and the loss of any aqueous being carefully avoided in the manipulation.

Third Stage.—The pressure of the vitreous will now cause the edges of the incision to bulge open into a long oval (Fig. 363) through which the knife-blade is raised upward, until above the iris-membrane, and then swung across the anterior chamber to a corresponding point on the right of the vertical plane, which, owing to the disturbance in the relation of the parts made by the first cut, is somewhat displaced and the second puncture must be made at least 1 mm. farther over—*i. e.*, 4 mm. to the right of the vertical plane (Fig. 363).



FIG. 362.—Plan of first incision.



FIG. 363.—First incision completed. Plan of second incision.



FIG. 364.—Pupil resulting from V-shaped iridotomy.

Fourth Stage.—With the knife-point again resting on the membrane, a second puncture is made by the same quick thrust, and the incision rapidly carried forward by the sawing movement to meet the extremity of the first incision, at the apex of the triangle, thus making a *converging* V-shaped cut (Fig. 364). Care must be taken at this point that the pressure of the knife edge on the tissue shall be most gentle, and that the second incision shall terminate a trifle inside the extremity of the first, in order that the last fiber may be severed and thus allow the apex of the flap to fall down behind the lower part of the iris-membrane. If the flap does not roll back of its own accord it may be pushed downward with the point of the knife.

When the operation is completed the knife is again turned on the flat and quickly withdrawn.[1]

Transfixion of the Iris in Iris Bombé.—This operation, designed and recommended by Fuchs, is performed as follows:

A narrow Graefe cataract knife is entered on the horizontal meridian 1 mm. to the inner side of the temporal margin of the cornea and passed through the anterior chamber to emerge on the opposite side at a point corresponding to the wound of entrance. As the iris is bulged forward in a roll around the pupil (see Fig. 150) the knife penetrates it both on the temporal and the nasal side and makes two holes in it at each of these situations. These holes do not close; the anterior and posterior chambers are again in communication and the tension falls. Should it be required an iridectomy can be performed later with far greater ease than would have been possible prior to the transfixion.

Division of Anterior Synechiæ (*W. Lang's Operation*).—This operation is performed with a pair of knives closely resembling Knapp's discission knife-needle. The one is sharp and the other blunt pointed. First, the sharp-pointed instrument is entered through the corneal tissue at a point favorably located for giving a fair lateral movement. It is next withdrawn, and the blunt-pointed knife passed through the same opening across the anterior chamber, with its cutting edge in contact with the synechiæ, which by means of a slight sweeping movement are divided. Occasionally the iris stretches so freely that it is difficult to sever it. Practically no reaction follows the operation, and the subsequent treatment consists in the use of atropin and a compress bandage. If it has been successful, the iris may be dilated and the distorted pupil become round.

This operation, according to Lang, is suited to adhesion of the iris or capsule to the wound after cataract extraction, to traumatic prolapses where a broad width of iris is clamped in the scar, to small adhesions due to perforating wounds or ulcers, and, finally, to large adherent leukomas. It is a useful operation, but in the last condition the effects are the least satisfactory.

OPERATIONS UPON THE SCLERA

Sclerotomy (*Anterior Sclerotomy*).—This is an operation first performed by Quaglino, and improved and advocated by de Wecker, which is practised for the relief of glaucoma, and by some surgeons is made to substitute the operation of iridectomy (see page 726). It is especially recommended in chronic glaucoma with deep anterior chamber, in inflammatory glaucoma with atrophy of the iris, and where iridectomy fails to reduce tension or to relieve the pain of old, blind, glaucomatous eyes. It is performed as follows:

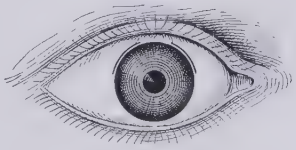


Fig. 365.—Lines of incision in sclerotomy.

A narrow Graefe's cataract knife, or a specially constructed knife known as a *sclerotome,* is passed through the sclera, 1 mm. from the margin of the clear cornea in front of the iris, and brought out at a corresponding point on the other side—*i. e.*, the puncture and counterpuncture are placed as if the surgeon intended to form a flap 2 to 2.5 mm. in height out of the upper (or lower) part of the cornea. The puncture and counterpuncture are enlarged with a slight sawing movement of the knife, which is slowly withdrawn before the section is complete, leaving the central

[1] The description of this operation is in Dr. Ziegler's own words, and the figures which illustrate it are his, and have been kindly loaned for reproduction.

quarter of the sclerotic flap, and as much of the conjunctiva as possible, except where punctured, undivided. Thus, at the upper (or lower) margin of the cornea there remains a bridge formed of sclera which connects the parts below it. If prolapse of the iris occurs, replacement should be attempted with a horn spatula. In the event of failure the prolapsed iris must be excised and the sclerotomy converted into an iridectomy. Preceding the operation eserin should be used to contract the pupil, and this drug must be continued during the process of healing.

Posterior Sclerotomy.—This is performed by entering a Graefe cataract knife at a point between the external and inferior recti muscles, 8 mm. from the corneal margin, and passing the blade through the sclera toward the center of the eyeball to a depth of 4 to 6 mm. As the knife is slowly withdrawn it is made to execute a quarter turn, the effect being the formation of a slight triangular wound, which favors filtration. The operation is employed in hemorrhagic glaucoma, preliminary to iridectomy (see page 437), especially when the anterior chamber is very shallow, and in retinal detachment (see page 516).

Internal sclerotomy, practised by de Wecker and by de Vincentiis, was designed to incise the tissue of the angle of the iris. The incision is similar to anterior sclerotomy, with omission of the counterpuncture, in place of which the arches of the pectinate ligament are incised.

Combined Iridectomy and Sclerectomy (*Lagrange's Operation*).—The operation, according to Lagrange, is performed as follows:

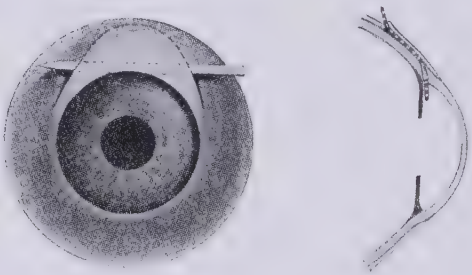


Fig. 366.—Lagrange's operation: Section of the sclera and conjunctiva (Lagrange).

With a Graefe knife the sclera is punctured at the outer side 1 mm. from the limbus and the counterpuncture is made at a corresponding point. The sclera is divided in the iridocorneal angle and the section includes the upper fourth of the cornea. In terminating the incision the cutting edge of the blade is directed backward in such a way as to bevel the sclera, and when the knife is beneath the conjunctiva a conjunctival flap (about 4 mm. in height) is made. In the second stage of the operation the conjunctival flap is raised, but not cut in any way, and drawn back on the cornea. This maneuver tilts the edge or tongue of the scleral flap upward. This is next removed with a sharp curved pair of scissors, and a sufficiently large piece of the sclera is resected from the exterior lip of the incision. Finally, iridectomy is performed in the usual way, and the flap of conjunctiva detached in the first stage of the operation is replaced, thus covering the defect in the sclera. The steps of the operation are evident by an examination of Figs. 366 to 368 borrowed from Lagrange's original paper.

It is not necessary to make a very large flap, 4 to 5 mm. is sufficient. Weeks, as the result of a large experience with this operation, recommends that it shall be performed as described by Lagrange, except that the incision shall be not more than 5 mm. (Lagrange advised 7 mm.). The shorter incision obviates the danger of prolapse of the ciliary body or of the lens into the wound and lessens the chance of escape of vitreous. The after-treatment should include, beginning forty-eight

hours after the operation, massage of the eyeball, which may continue for some days or even weeks according to the conditions (Weeks).

By means of this sclerecto-iridectomy a communication is made between chambers of the eye and the perichoroidal space and the subconjunctival cellular tissue. The hypertension of the glaucomatous eye, Lagrange maintains, is permanently relieved by the establishment

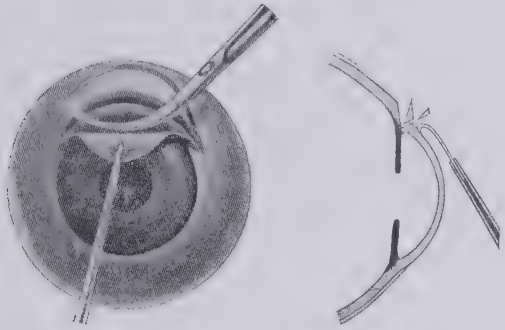


FIG. 367.—Lagrange's operation: Resection of the sclera (Lagrange).

of a fistulous track between the anterior chamber and the subconjunctival tissue. Iridectomy, he is willing to admit, cures acute glaucoma, but not the chronic variety. *Simple sclerectomy*, as practised by Lagrange, is performed in precisely the same way; that is, the first two steps of the operation are taken, iridectomy being omitted.

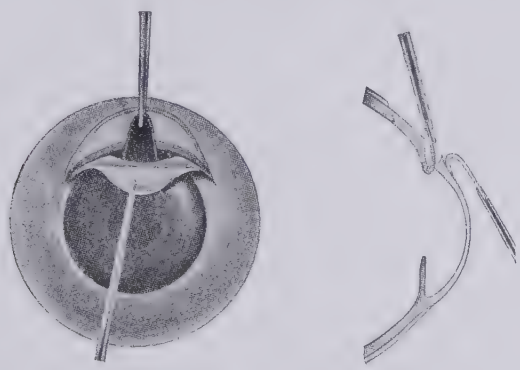


FIG. 368.—Lagrange's operation: Making the iridectomy (Lagrange).

Wedge-isolation Operation (*Herbert's Operation*).—H. Herbert has devised an operation which, he is satisfied, produces a permeable scar and which he describes as follows:

"The aim is usually to separate a sclerocorneal strip 6 or 7 mm. long. Subconjunctival puncture and counterpuncture are made with a tapering glaucoma knife the cutting edge being directed exactly upward, or inclined slightly backward. If a

small basal iridectomy is to be added, as it must be at times with an imperfectly contracted pupil, the point must come through the conjunctiva at a little distance beyond the scleral counterpuncture.

"An upward cut is then made in the sclerotic coat by sawing movements, as if to make a short scleral flap, but care is taken to avoid completing the section. This defines the posterior limit of the wedge. The blade is then brought down into the anterior chamber and an incision made forward *completely through* the sclera, at right angles to its surface. This forms the lower boundary of the strip of tissue. This incision is made slowly and carefully to avoid cutting the conjunctiva.

"The knife is then again passed up behind the partly isolated tissue, and brought forward to complete the separation. Only quite a narrow strip is isolated. Its exact outline in cross-section varies considerably throughout its length, and is probably a matter of little importance, provided that a fair proportion of the wedge is deep enough to reach the anterior chamber.

"If a small basal excision of iris is to be made, a conjunctival flap is cut sloping outward, but incomplete above. During the cutting the conjunctiva is pulled forward above the wedge with fixation forceps. But the writer (H. Herbert) now prefers generally to leave the iris intact. Deep impaction of the iris is avoided by keeping the pupil fully contracted during the operation and for the first day. Eserin drops, 1 per cent., are used, as required, beforehand; perchlorid irrigation of the conjunctiva is usually omitted; only three or four cocain instillations are made; and adrenalin is used once only, at the end, or omitted altogether. It may be well to displace the strip of tissue a little forwards, always before withdrawing the knife."

This technic has been modified in various ways: thus, the primary incision may be made with a bent keratome and the lateral cuts with a short narrow knife. N. Bishop Harman has devised "twin scissors" for the purpose of forming the lateral incisions.

Sclerectomy with Punch Forceps (*Holth's Operation*).—With a Graefe knife puncture and counterpuncture are made in the sclera 8 mm. from each other and 1 mm. from the limbus. Next, a scleral flap 2.5 mm. high and a conjunctival flap extending to 8 mm. from the limbus are formed. The conjunctiva is now freed from the anterior lip of the scleral flap, raised with forceps, and a piece of the scleral flap, 3 by 1.5 mm., is removed by means of the punch forceps (Holth-Vaclier's, de Lapersonne's, or Ziegler's instrument may be used). A basal or complete iridectomy may be performed, after which the conjunctiva is replaced. The primary incision may be made with a keratome which pierces the conjunctiva 10 mm. above the cornea, enters the sclera 2.5 mm. from the limbus, and penetrates obliquely into the anterior chamber. Holth also operates by making a conjunctival flap, followed by keratome incision 4 mm. long into the anterior chamber and excising a 3 mm. long piece of the sclera with his punch forceps; a basal or complete iridectomy concludes the operation. The conjunctival flap is replaced and held by stitches in the usual manner.

The important manner in which the results of these operations differ from cystoid cicatrices (see page 438) is that the scar is free from adhesion to the uveal tract. Some difference of opinion exists with respect to their permanent value, and Henderson, although willing to admit that at an early stage such a scar as has been described may be permeable and that filtration may take place, believes it soon becomes impermeable, owing to the ingrowth of epithelium, and, therefore, unless a fistula is made, filtration ceases. Lagrange believes that in his operation the fistulous track, before mentioned, is formed. The author has not had experience with Herbert's operation, but has performed the Lagrange operation with satisfaction in cases of chronic (simple) glaucoma. He has not employed it in cases of acute glaucoma, in which form of the disease a well-placed peripheral iridectomy has usually yielded excel-

lent results. Weeks recommends Lagrange's operation, except in buphthalmos, some types of glaucoma with deep anterior chamber, and cases of chronic glaucoma with relatively low hypertension. According to Meller, the delay in re-formation of the anterior chamber, which is noticeable in some eyes after Lagrange's operation, is due to detachment of the choroid. He regards the operation as unsatisfactory if the intra-ocular tension is very high. He reports 1.3 per cent. of *late infections* in 389 Lagrange operations. Intra-ocular hemorrhage, iritis, prolapse of the ciliary body, and loss of vitreous have been noted as complications.

Sclerectomy with a Trephine.—Trephining the sclera for the purpose of relieving the increased intra-ocular tension of glaucoma is not a new procedure. Long ago it was proposed by de Wecker, Argyll-Robertson, Strawbridge, Blanco, and Froelich, but their operations differed in many respects from the newer procedures of scleral trephining which have been suggested.

Scleral Trephining (*Fergus' Operation*).—The technic is thus summarized by Ballantyne: A conjunctival flap is dissected up toward the cornea and laid over the corneal surface, while with a Bowman's trephine a small disk of sclera is removed 1 or 2 mm. from the apparent corneal margin. Next, an iris repositor is passed from the trephine opening into the anterior chamber, keeping it in close contact with the sclera and cornea. The conjunctiva is then replaced and stitched into position.

Sclerocorneal Trephining (*Elliot's Operation*).—The description of the technic of this operation, which has justly achieved a world-wide reputation, at the author's request has been written by Lieut.-Colonel Elliot.

1. **The Quadrant of the Eye Selected.**—This should invariably be the upper unless there is some strong contra-indication. The presence of an upward coloboma resulting from a previous iridectomy does not contra-indicate an upward trephining; on the contrary, the operation is made easier thereby.

2. **The Flap.**—This should be large. The incision runs roughly concentric with the limbus and ends on either side opposite the highest point of the cornea, and about 8 mm. from its inner and outer sides. The conjunctiva should be seized as high as possible with forceps and drawn well down, while the patient looks strongly toward his feet. One free horizontal cut, followed by a couple of snips at each side, will outline the flap. Only the central area of the flap thus marked is to be dissected up. As we approach the limbus, we should work down to the sclera and should lay the latter bare in the last few millimeters of the wound. We must then clearly define the limbus as a rounded ridge overhanging the adjacent sclera. The area for the application of the trephine must be cleared of all tags of loose tissue. The conjunctival flap should be gently drawn downward by the aid of a blunt instrument, and the cornea is then to be split with the scissor points or with any other fairly sharp instrument. As the dissection proceeds the "dark crescent" of the cornea can be seen as an area convex in outline toward the sclera, and with a straight edge on the corneal side. It is sufficient to split the cornea over an area 1 mm. in depth. This enables a 2-mm. trephine to be applied half on the cornea and half on the sclera.

3. **The Application of the Trephine.**—The trephine (2 mm. in diameter) should be placed *as far forward as possible*, being slid into place from the scleral side, the edge of the flap being meantime keenly watched, so as to avoid buttonholing. A

sharp blade is required to make sure of cutting a definite groove in the corneosclera on its first application. Our object should be to make the blade cut through first on its corneal edge, and to ensure this we must slope the upper edge of the instru-

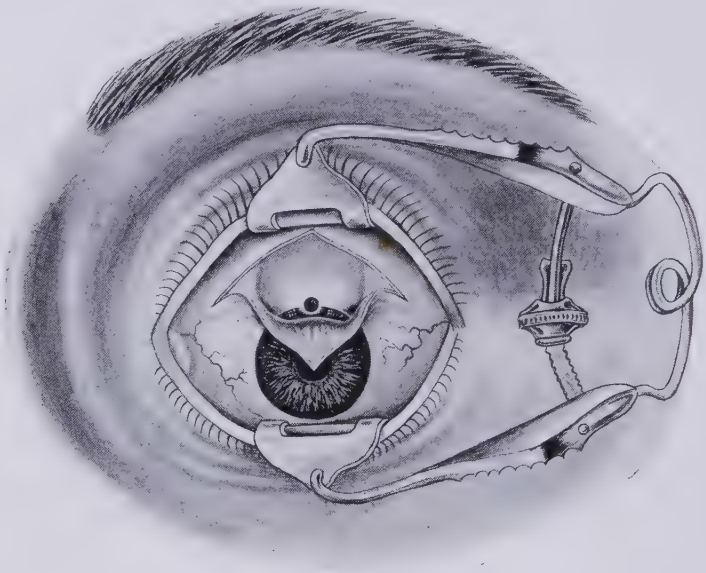


FIG. 369.—Sclerocorneal trephining: Conjunctival flap reflected; trephine opening partly in the cornea and partly in the sclera. (After S. Lewis Ziegler.)

ment a little toward the patient's feet, so that the disk cut out may be hinged on its scleral side. A bead of iris tissue will prolapse through the corneal side of the hole, pushing the disk before it. The disk together with the prolapsed iris is seized in a

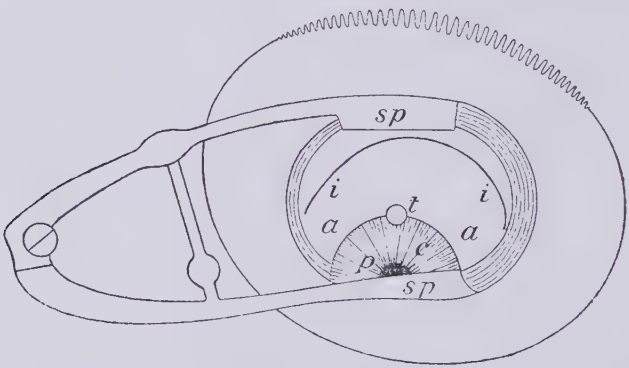


FIG. 370.—*sp*, Speculum; *i*, *i*, incision; *c*, cornea; *t*, trephine hole; *p*, pupil; *a*, *a*, channels in conjunctiva along which filtration fluid passes to enter the main area of the subconjunctival space. (Elliot.)

pair of iris forceps and the two structures are cut with one snip of the scissors, thus performing a sclerecto-iridectomy with a single cut. The amount of disk removed can be graduated according to the needs of the case.

4. **Toilet of Wound.**—The iris is replaced by massage, aided by an irrigator, if necessary, and a dressing is applied. The rôle of the iridectomy is the same here as it is in cataract extraction; no more, no less. It is safer, in European (and American) practice, to secure the flap by means of one or two sutures.

5. **Instillation of Drops.**—From the second or third day onward, provided the tension is down, atropin drops are freely instilled.

6. **After-treatment.**—The unoperated eye is opened after twenty-four hours, and both eyes after forty-eight hours. The patient sits up in bed on the second day and moves about the room on the third day.

Naturally, various operators have advised modifications of the procedure; for example, L. Webster Fox (who prefers his type of the von Hippel trephine) forms the conjunctival flap as in the Van Lint sliding flap in cataract extraction, and McReynolds dissects out a piece of sclerocornea with a knife-point after fixing it with a traction suture. In the opinion of the author the most satisfactory results are obtained if Col. Elliot's technic is exactly followed. The author prefers a hand-driven trephine; good models are those designed by Elliot, Stephenson, Lang, and Cross.

Indications.—Although many surgeons utilize this operation in acute glaucoma, the author is unconvinced that it is a better procedure than a technically correct iridectomy. It and other methods which secure a so-called "filtering area" are better operations in chronic, non-congestive glaucoma than simple iridectomy. If an iridectomy in this disease has failed of its purpose, corneoscleral trephining is preferable to a second iridectomy or sclerotomy. In chronic glaucoma with greatly contracted field it is a safer operation than iridectomy. It is not a wise procedure if glaucoma is complicated with cataract, but it should be considered in glaucoma secondary to cataract in those eyes in which the vitreous and aqueous chambers are not in communication. It may be performed in absolute glaucoma in the hope of preventing enucleation, but this prevention is not assured. It is not likely to meet with success in glaucoma due to intra-ocular hemorrhage and thrombosis of the central retinal veins; it may be tried in staphyloma and secondary glaucoma, but the outlook is not a brilliant one. In buphthalmos it has achieved satisfactory results (see also page 443). Colonel Elliot recommends his operation in all cases of glaucoma except those which are secondary to cataract extraction where vitreous is in the anterior chamber. Its presence in the chamber can be detected by the slit lamp (page 478).

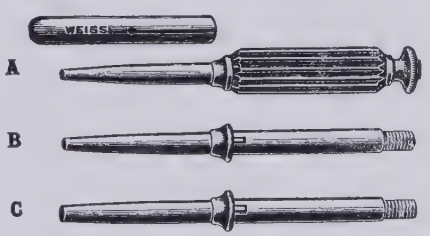


FIG. 371.—Stephenson's sclerectomy trephines.

Complications and Causes of Failure.—The chief complications are buttonholing the conjunctival flap, extensive hemorrhage into the anterior chamber, entrance of the scleral button into the aqueous chamber, wounding the lens, loss of vitreous (which should never occur if the

trephine opening is correctly placed); intra-ocular hemorrhage, detachment of the choroid, purulent infection, and iritis. After this operation iritis occurs not infrequently, and appears early either as a *quiet iritis*, with almost no signs of inflammation of the uveal tract, but with the gradual development of soft synechiæ, or arises as a sharp, plastic inflammation at a later period than the first type of the affection. Walter Parker's investigations indicate that iritis is much less apt to develop if complete iridectomy is part of the operative procedure—a significant observation. Its incidence should be prevented by the early use of a mydriatic—the author prefers scopolamin. Occasionally, the anterior chamber is re-established slowly and a week or more may elapse before it is reformed. This may be due to detachment of the choroid which almost always subsides, or to failure in even healing of the conjunctival incision. In the latter circumstances the line of incision should be touched with a 2 per cent. solution of nitrate of silver.

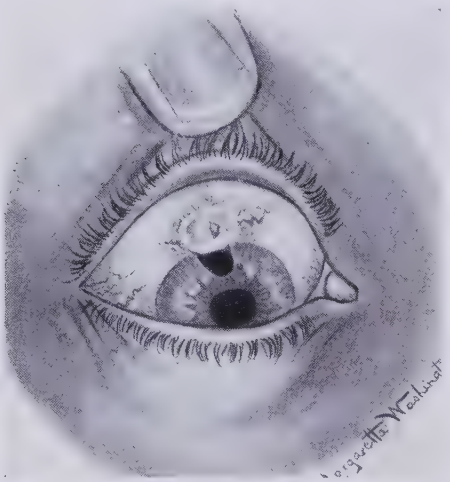


Fig. 372.—Corneoscleral trephining: Note the filtering area at corneoscleral margin; small peripheral iridectomy.

The causes of failure in this operation, according to Elliot, depend upon forward dislocation of the lens or vitreous body and prolapse of uveal tissue into the trephine hole, which is blocked by proliferated connective tissue either from the episclera or the uvea. In Stephenson's observations the trephine tract was occluded by vascular fibronuclear tissue and iris pigment. Treacher Collins ascribes failure to leaving behind Descemet's membrane, to local infection and the formation of granulation tissue, and to a down growth of epithelium from the surface between the lips of the wound. A number of *late infections* (from a few weeks to some months after operation) are now on record, the process varying in severity from an infected iridocyclitis and hypopyon to panophthalmitis. This disaster is chiefly to be feared if the conjunctival flap is too thin; it should be formed of a proper thickness. Late infections not peculiar, however, to this operation (see page 737) are due to the entrance of micro-organisms through delicate and invisible fistulas in the conjunctiva covering the trephine opening. After this operation patients with very thinly covered "filtering areas" should be carefully watched and should daily use a collyrium of boric acid and sulphate of zinc (Gifford). Fluorescein should be employed to detect the presence of epithelial defects (Axenfeld, Harms). Pronounced hypotony, as it may occur after this operation, A. Knapp regards as a dangerous

complication, and he advises a small trephine and removal of a small disk.

Iridotasis (*Borthen's Operation*).—This operation, devised by Borthen, has many advocates, and in this country is especially commended by David Harrower and W. H. Wilder. The method of operating, according to Harrower, who essentially follows Borthen's technic, is as follows:

> Fifteen minutes prior to operation a drop of a 1 per cent. solution of atropin is instilled. Cocain anesthesia follows. Next, the conjunctiva being grasped with forceps 10 mm. back from the cornea, an incision, 10 to 12 mm. in length parallel with the corneal line, is made in this membrane which is then separated from the sclera to the corneoscleral junction. Finally, an incision, 4 mm. wide, is made (Borthen uses a special knife, having a shoulder, which regulates the depth of penetration) just behind the corneal margin, through which a forceps is introduced, the iris grasped, withdrawn into the scleral opening, and the conjunctiva smoothly replaced over it. If successful a satisfactory "filtration bleb" results.
>
> Wilder, who commends the operation, makes his conjunctival flap as if for Elliot's operation (page 737), and insists that the pull on the iris must be a gentle one and that the posterior pigment-layer surface must be exposed. He does not recommend a preliminary instillation of atropin, but employs this drug on the day following the operation.

Complications are uncommon and healing is usually prompt, but late infections have been recorded (Dunbar Roy; also one by Wilder, fifteen months after a successful operation) and return of high tension has been reported (A. Knapp). The operation is advised in chronic glaucoma, but it has also been utilized in acute glaucoma. In Holth's *iridencleisis* the incised iris is incarcerated in the scleral keratome-incision and the conjunctival flap allowed to heal over it.

Zorab's operation consists essentially of thread drainage of the anterior chamber. A silk thread is introduced into the aqueous chamber and covered with a conjunctival flap. Casey Wood, in this country, has practised this procedure.

E. J. Curran, believing that the flow of aqueous from the posterior to the anterior chamber is impeded in glaucoma "on account of the iris hugging the lens over too great a surface," has designed an operation the purpose of which is to establish a drain from the posterior to the anterior chamber. It consists essentially in cutting with a delicate knife-needle a 1-mm. hole in the upper surface of the iris.

Cyclodialysis (*Heine's Operation*).—By means of this operation, suggested by Heine in 1905, an endeavor is made to form an artificial communication between the anterior chamber and the suprachoroidal space, but there is no positive proof that after it drainage occurs into this space, although it is possible that a successful cyclodialysis reopens the angle and brings it again into communication with Schlemm's canal. It is performed as follows:

> After the reflection of a small conjunctival flap, preferably on the outer side of the eyeball, an opening is made into the sclera with a straight lance, parallel to the corneal margin and from 6 to 8 mm. away from it, without injuring the uveal tissue. This opening should be from 2 to 3 mm. in length, and through it a spatula is intro-

duced with which the ciliary body is separated from the overlying sclera and the instrument gradually pushed through the ligamentum pectinatum into the anterior chamber. Finally, a quadrant of the iris periphery is detached. Occasionally some difficulty is experienced in passing the spatula between the ciliary body and the sclera into the anterior chamber, and in a few instances hemorrhage into this chamber has occurred.

If the aqueous escapes the intra-ocular tension is immediately reduced, but, according to Meller, in successful cases reduction of tension is not noticeable until the following day. All increased tension should disappear by the second or, at the latest, by the third day. Occasionally subnormal tension results. If the tension remains low for a week, the ultimate result is likely to be favorable. Meller states that permanent good results do not exceed 20 per cent. The operation has proved to be satisfactory in chronic glaucoma, in secondary glaucoma due to anterior synechia or subluxation of the lens, in glaucoma following cataract extraction, in return of tension following a Lagrange or Elliot operation, in cases of chronic glaucoma where iridectomy has failed, and in absolute glaucoma. It is contra-indicated in acute glaucoma and in glaucoma of an exudative type (H. S. Gradle). Complications which may arise are detachment of Descemet's membrane which produces a temporary opacity of the cornea, hemorrhage as noted above, and iridodialysis, which is really not disadvantageous; acute rise of tension has been reported. The author has frequently performed this operation and some of the results have been permanently good. It may be repeated several times if the high tension returns. It is not an operation which can replace iridectomy or other well-established procedures, but it may be used to lower ocular pressure and be followed later by iridectomy. Shallowness of the anterior chamber is no contra-indication.

Operations for Detachment of the Retina.—Some of the means devised for the cure of retinal detachment from the operative standpoint have been referred to on page 516. Three additional procedures are the following:

1. **Trephining the Sclera for Detachment of the Retina.**—Attention to this operation in this respect was first prominently called by Walter R. Parker:

His technic consists essentially, after raising a suitable flap of conjunctiva, in making a 2-mm. trephine opening in the sclera at a point corresponding to the lowest portion of the detached retina. Usually, there is a free escape of subretinal fluid, followed sometimes by an escape of a few drops of vitreous. The conjunctival flap being replaced and secured by a single suture, the patient is required to rest in bed for ten days in such a position as to favor replacement of the retina.

Edgar Thomson and T. H. Curtin, using a 2 or 3 mm. trephine and selecting for the site of operation the most dependent position which is possible within the area of detachment, after the escape of the suprachoroidal fluid and replacement of the conjunctival flap keep the patient in bed (pupil dilated and bandage applied) for ten days. At the expiration of this period the needle of a small aspirating syringe is inserted into the subretinal space through the conjunctiva and choroid and the fluid forcibly aspirated. This aspiration may be repeated, if necessary, and may be performed immediately on completion of the trephining.

2. **Sclerotomy Combined with Electrolytic Punctures** (*Verhoeff's Method*):

Verhoeff first performs posterior sclerotomy in the usual manner, and keeps the patient in bed with both eyes bandaged for a week. At the conclusion of this preparatory treatment, the object of which is to bring the retina in contact with the choroid, he makes a large number of minute punctures through the sclera and retina by electrolysis, employing a small steel, half-curved eye needle, the current being obtained from a series of six dry batteries of 1½ volts each, the positive electrode being applied by means of a wet sponge to the cheek. The needle point is pressed firmly against the globe until it penetrates the ball, when it is pulled back slightly, so that the point protrudes only a millimeter or two through the vitreous, where it is allowed to remain for about five seconds. The number of punctures must vary according to the extent of the detachment.

3. **Resection of the Sclera** (*Müller's Method*).—This operation, devised by Müller, is advocated by Török, who describes it as follows:

The external rectus is exposed, two sutures inserted, and the muscle severed between them. An elliptic space, 20 mm. in length and 10 mm. in width, is outlined on the sclera, its anterior end behind the insertion of the muscle and its posterior border toward the equator. The elliptic incision is next made half-way through the sclera and five fine catgut sutures are inserted from within or within outward. The sutures being raised out of the way the posterior border of the incision is carried through the entire thickness of the sclera, the choroid separated from the sclera, the sutures drawn together whereby the scleral flap is pushed into the pocket between the sclera and the choroid; prior to tying the last suture the choroid is punctured. The divided muscle is sutured in place, the conjunctival wound closed, atropin is instilled, and the patient required to remain in bed in a prone position for eight days.

Successes have been reported with all of these operations in the sense that vision was improved and the field widened or restored, sometimes only temporarily and sometimes long enduring. In some instances the operations have been failures and in others conditions have been worse after their performance. The author has had no experience with Verhoeff's method nor with Müller's resection. His best results have been secured with posterior sclerotomy, followed by subconjunctival injections of physiologic salt solution or sodium citrate (4 to 5 per cent. diluted with 2 to 4 parts of sterile physiologic salt solution), rest in bed and locally dionin and atropin; as adjuncts diaphoresis and diuresis have also been employed. On the whole, with a few exceptions, the permanent results have been disappointing. Certainly, in general terms, the history of the results of operations in detachment of the retina is not a brilliant chapter.

OPERATIONS ON THE GLOBE AND REMOVAL OF FOREIGN BODIES

Enucleation of the Eyeball.—The following instruments are necessary: A stop speculum, fixation forceps, dissecting forceps, strabismus hook, and a pair of scissors curved on the flat (enucleation scissors):

The lids are held apart by a stop speculum while the surgeon divides the conjunctiva and adjacent fascia with scissors in a circle as close as possible to the mar-

gin of the cornea. This is sometimes called "circumcising the cornea." At the same time the anterior attachments of Tenon's capsule are divided, thus opening Tenon's space. The tendons of the ocular muscles, beginning with the superior rectus, are next successively raised upon a strabismus hook and divided. The eye being made to start forward by inserting the stop speculum somewhat more deeply, the eye is drawn forward, the face of the patient being turned toward the operator, and the curved scissors are introduced on the nasal side between the severed conjunctiva and the freed eyeball, and made to follow the curve of the latter until the optic nerve is reached, where the blades are expanded and the nerve seized and cut squarely off. The attachments of the oblique muscles and the remaining tissue which may cling to the eyeball are then severed. Subsequently the conjunctival wound is closed with a few interrupted sutures.

Hemorrhage is usually not severe, and is readily controlled by pressure. After freely irrigating the socket with a bichlorid solution a full aseptic dressing should be applied. In place of general anesthesia, local anesthesia, by means of retrobulbar injections (see page 690), is preferred by some surgeons.

The operation just described is sometimes known as Bonnet's method. The eye may also be rapidly removed according to the Vienna method:

Either the tendon of the external or the internal rectus is first divided. Next, the eyeball being steadied with a forceps which grasps the stump of the tendon, the other recti muscle insertions are severed with scissors, without being caught up on a strabismus hook. Finally, the optic nerve is divided in the usual way and the attachments of the oblique muscles severed.

The technic of enucleation, however, has been materially improved, chiefly by the various methods of suturing the tendons to the conjunctival bed to prevent their retraction. Suker sutures the severed ends of the recti muscles one to the other, after which the conjunctiva from above and below is brought over the muscle-stump and fastened with a continuous suture, which also attaches the conjunctival covering to the muscle-stump. H. Schmidt secures each rectus tendon with a catgut suture and makes a slit in the conjunctiva over each muscle, in which the divided conjunctiva is fastened. The conjunctiva is brought together with a continuous suture. Priestley Smith pinches up a narrow horizontal fold of the conjunctiva over the internal rectus, so as to include the subjacent connective tissue and muscle, and carries a black silk suture through these structures with a curved needle, the suture being tied firmly, but not too tightly. In a similar manner the other straight muscles are attached, after which the enucleation is carried out in the usual manner and the conjunctival aperture closed with one or more vertical sutures. Frederic Krauss has devised a more elaborate procedure whereby the relations of the ocular muscles to Tenon's capsule are preserved and the rotations of the stump increased.

Freeland Fergus' method of enucleation is as follows:

The conjunctiva is divided freely over the external rectus so as to expose thoroughly that muscle. As soon as this is done, one blade of the scissors is passed beneath the muscle and it is divided, a small portion of its tendon being left attached to the sclera. This portion is taken hold of with the forceps and the eye is gently rotated toward the inner canthus. When in this position the optic nerve is severed.

After the division of the nerve the movement of rotation is continued and all the other tissues are, as they come in view, resected as closely as possible to the sclera. A few snips of the scissors suffice. The wound, which is always neat, may be closed with or without the insertion of a gold ball, or with or without suturing together of the muscles, as recommended in the following paragraph.

The author operates in the following manner with satisfactory results:

The conjunctiva is divided as close as possible to the corneal margin; each rectus tendon is next seized with forceps, separated from the sclera, and drawn forward to the edge of the cut conjunctiva, where it is fastened with a black silk suture. The eyeball is next enucleated in the ordinary manner, hemorrhage being checked by packing the cavity with a small wad of sterilized gauze. Finally, after removal of the packing, the edges of the conjunctiva are united with interrupted sutures which are generally placed in a horizontal direction, and which also include the capsule of Tenon. The usual dressing is applied, both eyes being bandaged for twenty-four hours.

The effect of this operation, whereby each rectus tendon is "advanced" to the margin of the conjunctiva and prevented from retracting, is to give a movement to the conjunctival bed very much greater than that which is secured after the ordinary enucleation.



FIG. 373.—Average artificial eye or shell.



FIG. 374.—Solid artificial eye.

Accidents.—(*a*) *Hemorrhage.*—Occasionally severe hemorrhage occurs during the enucleation of an eyeball, sometimes caused by an anomalous distribution of the vessels. If necessary, the orbit can be packed with antiseptic gauze. The tissues of the orbit may become very much infiltrated with blood and puff out in an alarming manner. The blood-clot, however, is usually absorbed, and no harm results, although a hematoma of the orbit so pronounced as by pressure to threaten the integrity of lids has been reported.

(*b*) *Perforation of the Sclera.*—Sometimes, especially in a ball having very thin walls, the sclera is punctured in the endeavor to cut the optic nerve. This simply complicates the operation, because it is more difficult to remove a collapsed ball than one which is distended. Should the operator be so unfortunate as to cut through the sclera and leave a portion of it remaining behind, he must proceed to search for the fragment, which can be picked up with forceps, and cut it off, together with the nerve.

(*c*) *Consecutive or Secondary Hemorrhage.*—Occasionally a consecutive or secondary hemorrhage occurs after enucleation. The bandages should be removed, the lids separated, the blood-clot removed, the orbit irrigated with an antiseptic fluid, and, if pressure fails to stop the

hemorrhage, a packing of antiseptic gauze should be inserted. Excessive hemorrhage in hemophilic subjects has been checked by the intravenous injection of normal blood-serum.

The **after-treatment** of an enucleation consists in placing the patient in bed, certainly for the first few days. No severe pain ought to follow an enucleation, and decided headache, elevation of temperature, and restlessness may indicate meningeal complication. In a certain number of instances meningitis has followed the operation, especially when it has been performed on an eye within which suppuration is taking place. Under modern methods of operating and with antiseptic precautions this accident is, fortunately, a rare one.

Insertion of Artificial Eyes.—An artificial eye may be inserted as early as the second or third week after an enucleation of the eye; indeed, some operators insert it at a much earlier date. For the first week or two the artificial eye should be smaller than that which is a perfect match for the opposite side. The eye may then be exchanged for one which in size is as nearly as possible a match for the fellow-eye. At first the eye may be worn for several hours at a time. Soon it can be worn all day, but it never should be allowed to remain in the socket during the night. It is not necessary to keep an artificial eye in water during the night. It should be washed with a little alcohol and water and allowed to dry.

In order to insert an artificial eye, the upper eyelid is seized between the fingers of the left hand and drawn gently down and out, and the larger end of the shell is inserted vertically beneath it, then brought to a horizontal direction, while at the same time the lower lid is pulled down, when the shell slips into place. In order to remove an artificial eye, the head of a large pin is inserted beneath its lower margin, the lower lid being at the same time depressed, while the eye is tipped upward and forward, when the pressure of the upper lid will force it out. Very soon patients become exceedingly expert in taking out and introducing artificial eyes, and do not require the aid of a pin in making the manipulation just described. Owing to failure to care properly for the socket it may become infected, with the development of much mucopurulent or purulent secretion. It is a troublesome condition. Bichlorid of mercury and boric acid lotions may be tried. If the pneumococcus is present, optochin (2–5 per cent.) and mercurophen (1 : 8000) are useful. *Dichloramin-T* in eucalyptol is excellent. Lawson's recommendation of *flavine* (proflavine) in septic wounds and some forms of purulent conjunctivitis suggests its trial in this condition, the strength of the solution being 1 : 1000.

One of the chief objections to the shell-shaped prosthesis, or artificial eye, is the fact that in its hollow under surface tears and mucus may accumulate, while its thin edges may bruise the conjunctival bed. To obviate this difficulty the so-called "reformed artificial eye" has been introduced, largely through the efforts of Professor Snellen, which consists of a double-walled shell, or sometimes of a solid eye, the smooth rounded contour of which neutralizes the objections to the thin edges

of the old-fashioned shells. The movements of an eye of this character, placed in the socket after a properly performed enucleation with suture of the tendons or implantation of a gold sphere in Tenon's capsule, are nearly as extensive as those which follow Mules' operation.

Instead of the operation of enucleation, certain substitutes have been proposed, the most important of which are:

Evisceration of the Eyeball.—This consists in an evacuation of the contents of the eye, the sclera being unmolested, and closure of the scleroconjunctival wound with sutures, thus forming a movable stump for the artificial eye.

The instruments required for the operation are a speculum, fixation forceps, a narrow knife, a pair of scissors, and an evisceration spoon. It is performed as follows:

> The speculum being introduced, the conjunctiva is loosened around the cornea; the anterior chamber is transfixed with the knife on a level with the horizontal meridian, the lower portion of the cornea separated, the flap seized with forceps, and the remainder of the cornea cut away at the corneoscleral margin. With the evisceration scoop the contents of the globe are thoroughly and cleanly evacuated. The cavity of the globe is wiped out with sterilized cotton-wool, and all bleeding is stopped. The edges of the conjunctiva are united by means of a suture similar to the string which draws shut a tobacco-pouch—a suture sometimes called the *tobacco-pouch suture*, or by interrupted sutures. These may include the conjunctiva alone, unless this is very much macerated, when it may be necessary to include the sclera. The author is accustomed to suture both the sclera and the conjunctiva. The evisceration may be accomplished without sacrificing the cornea provided this membrane is not infected (Gifford).

Considerable pain may follow the operation, together with edema and swelling of the surrounding tissues. In order to avoid this it has been recommended to introduce a horsehair drain, and Prince has suggested wiping out the cavity with dilute carbolic acid in order to allay the pain.

The chief indication for evisceration is panophthalmitis (see also page 401), although it may also meet the indications which are mentioned in connection with Mules' operation. Evisceration is contra-indicated by sympathetic inflammation or irritation, malignant disease, and much shrunken eyeballs. Although the stump after evisceration is primarily more voluminous than that which is secured after an enucleation, subsequent shrinking of this stump ultimately renders the cosmetic effect of the operation no better than that which is secured by a properly performed enucleation, while its inconveniences are much greater. If Gifford's plan is pursued (*simple evisceration*), that is, without keratectomy, the stump is more voluminous than after the ordinary evisceration.

As a substitute for evisceration Sir William Lister has advocated and practised the enucleation of a septic eye with this modification, namely, that a fringe of sclera about 10 mm. in width is allowed to remain and surround the optic nerve entrance, avoiding, therefore, opening the optic nerve sheath and the danger of conveying infection through this route.

Evisceration of the Eyeball, with Insertion of an Artificial Vitreous.—Mules' Operation.—Mules modified the operation of evisceration by the introduction of a glass ball into the cavity of the sclera. The operation is performed as follows:

After general anesthesia a stop speculum is introduced, and the conjunctiva dissected from the corneoscleral attachment in all directions to the equator of the ball without disturbing the muscles. The cornea and 1 mm. of the scleral margin are removed in the manner described under Evisceration. Next the contents of the globe are emptied by any convenient method, a small evisceration scoop being a satisfactory instrument. Great care must be taken to remove the entire contents, leaving a perfectly clean, white sclera. Hemorrhage is controlled by packing the scleral cavity with sterilized gauze, and by frequently irrigating it with a tepid solution of bichlorid of mercury (1 : 5000). A glass or gold sphere (gold is preferable), of such size that it may be introduced within the scleral cup without difficulty, is selected, its introduction being facilitated by slitting the sclera vertically for about 4 mm. at the upper and lower margins of the opening. The introduction of the sphere is further facilitated by the use of an instrument specially devised by Mules for this purpose. The concluding steps of the operation consist in stitching the sclera vertically, the conjunctiva horizontally, and applying a full antiseptic dressing. The patient should be confined to bed for at least four or five days. Considerable reaction may follow, and marked chemosis of the conjunctiva. This may be controlled by the continuous application of cold, and probably be avoided by not removing the bandage for forty-eight or even seventy-two hours. Mules recommended that the sutures should be of catgut; the author prefers silk sutures.

Victor Ray has modified this operation in that after evisceration of the scleral contents he removes from within a circular piece of the sclera, 20 mm. in diameter, which includes the entrance of the optic nerve and ciliary nerves and vessels and fills the cavity with fat instead of a gold ball. In like manner, Dimitry forms a posterior window in the sclera, but uses a gold ball as in the ordinary Mules operation.

The chief *indications* for this operation are ruptured or injured eyeballs, provided the sclera is not too much lacerated, and the accident of recent date; staphyloma of the cornea and sclera, or complete leukoma; absolute glaucoma, buphthalmos; and non-traumatic iridocyclitis. The chief *contra-indications* are suppuration of the eyeball; morbid growths; much shrunken eyeballs, the contents of which have undergone bony or calcareous change; sympathetic ophthalmitis, sympathetic irritation, and pathologic conditions of the eyeball which are likely to produce either of the last-named affections; extensive injuries of the eyeball, with much bruising and laceration of the sclera; dacryocystitis; and ocular conditions demanding enucleation or its equivalent in very old persons.

Implantation of an Artificial Globe in Tenon's Capsule After Removal of the Eyeball (*Frost-Lang Operation*):

The eyeball is enucleated in the manner already described, and, after all bleeding has been checked, a glass or gold (gold is preferable) ball is inserted within Tenon's capsule. The size of this ball should not be less than 14 mm. in diameter (Greenwood insists it should be 18–20 mm. in diameter) and the capsule should be carefully sewed over it with fine silk sutures. It is the practice of some surgeons to include the sphere within the grasp of the recti muscles by stitching the superior rectus to the inferior rectus by means of a mattress-suture and the lateral recti by a similar suture. This plan apparently possesses no material advantage, provided the

muscles are secured in the manner described. In place of interrupted stitches a purse-string suture may be used, the important point being that the capsule shall be sewed first over the ball and the conjunctiva over the ball thus enclosed. The conjunctival sutures may be placed in a transverse direction or vertically (Greenwood prefers the latter position). The suture line should be painted with a 5 per cent. solution of iodin or 1 : 1000 lotion of flavine (Lawson). The after-treatment does not differ from that accorded to an enucleation; the reaction is usually very slight. If great care is taken to apply the sutures (some surgeons prefer fine catgut) in the manner described, extrusion of the ball is most uncommon.

This operation may replace ordinary enucleation in all cases except where sympathetic ophthalmia is threatened or present or the eyeball is septic (panophthalmitis); it is usually not performed in the presence of an intra-ocular growth. The cosmetic results are excellent. Paraffin spheres have been recommended in place of glass or gold balls; they possess no advantage in this respect. Silver-coated balls must not be used; they undergo disintegration as the result of oxidation.

Implantation of Cartilage.—Many surgeons prefer natural tissues to metallic or glass prosthesis. Transferred cartilage establishes, it is said, fresh communications with the blood-vessels in its vicinity, and becomes fixed to the capsule in recent enucleations. Even if the cartilage after implantation is transformed into a species of fibrous tissue, it is believed that the cosmetic result is not disturbed.

For the purpose of making cartilage implantation generally the seventh or eighth rib is selected and the globe of cartilage removed with a trephine of such size as is suited to the conditions. The cartilage having been introduced within Tenon's capsule, it is sutured to the inside or, if possible, the four straight muscles are attached by means of sutures to this cartilage sphere; the rest of the operation proceeds as has already been described. It has been stated that such grafts may be used even in the presence of septic eyes or septic sockets. The author has no experience in this respect, but doubts the propriety of an implantation in a septic socket.

In place of human cartilage it is the practice of some surgeons, notably Magitot, to use formalized cartilage taken from a calf or lamb rib. This is placed in formol, 10 per cent., for three days, and afterward freed from the formol by successive washings in sterile water. It is most important that there shall be a thorough removal of all traces of the formol before the cartilage is implanted, which can be shaped to any size that is required for the purpose of the implantation.

Terrien recommends a graft of cartilage, generally rib cartilage, 1.5 cm. in length, to which the tendons of the rectus muscles are sutured. In default of the graft prosthesis he generally improved the appearances by using artificial stumps of hard ebonite shell in an envelope of soft India-rubber, which was molded to the bottom of the conjunctival sac, or molds of wax were placed behind the artificial eye.

Fat Implantation Into Tenon's Capsule.—This operation, originally proposed and practised by Barraquer, came into prominent notice after Bartels called attention to the operation in 1908.

The eyeball is enucleated, the recti muscles being secured in the manner already described. A mass of fat taken from the abdominal wall is inserted into Tenon's capsule so as to fill, but not to overstretch, it. The muscles are sutured crosswise or by means of a purse-string suture, and next the cut conjunctiva is united with stitches. Either catgut or silk may be employed. The cosmetic result is good.

A number of modifications of this operation have been suggested in the amount of fat to be inserted and in the management of the muscles. The mass of fat may be covered with fascia lata over which the muscles are secured in the usual manner. B. W. Key has devised an elaborate technic whereby he strives to obtain a healthy and permanent growth of fat within Tenon's capsule and to attach the muscles in such a manner that the stump shall have decided rotary motion.[1]

Implantation of a Glass or Gold Ball Into the Orbit After Remote Enucleation of an Eyeball.—L. Webster Fox operates as follows:

If the operation is to be performed on the right orbit, the eyelids are separated by a speculum, the conjunctiva is grasped up and in above the inner canthus, and the tissues are well pulled out. Next, a Beer's knife or curved keratome is passed through the tissues somewhat obliquely and well down into the orbit, and an opening made large enough for the insertion of the globe behind the tissues. This opening may be enlarged with curved scissors to the desired size. When ready, a gold ball is inserted through the opening, which is closed with two stitches and over which a shell is placed, modeled after an artificial eye. The eyelids are then closed over this shell, which is left in place for twenty-four hours. The stitches are taken out on the third day. If the operation is to be performed on the left orbit, the incision is made up and out above the external rectus muscle and the dissection carried out as above described. After healing, the artificial eye is inserted in the usual manner, and, naturally, the support of the implanted globe improves the cosmetic result. A comparatively large percentage of these balls escape, or are extruded; moreover, the sphere may leave its central position, causing the overlying glass eye to be turned in some eccentric direction.

To obviate this a method may be utilized as follows: The conjunctiva is incised in a horizontal direction and dissected from the underlying tissue. Tenon's capsule is identified and freed, and within its space the ball is placed. If possible the ocular muscles are found and secured in the manner already described, the whole procedure being a replica of a primary implantation.

In place of a gold or glass sphere cartilage may be used, or fat wrapped in fascia lata. The insertion of sponge-grafts, glass balls wrapped in a sponge-layer, or paraffin sphere have been recommended in primary and late implantations, but possess no advantage over those which have been described; as previously noted, silver balls are not advisable, as they undergo oxidation within the tissues.

Opticociliary neurotomy and neurectomy have been employed as substitutes for enucleation, and are still performed by some surgeons, but in the opinion of the author they are rarely to be recommended.

Removal of Metallic Foreign Bodies from the Interior of the Eye.—For this purpose, as has already been recorded on page 331, a giant magnet may be employed (the Haab pattern), and the body drawn into the anterior chamber, or, having been properly localized by means of the *x*-rays, it is removed by means of a large magnet (the Sweet model, Lancaster model), through a suitably placed scleral incision which directly overlies the position of the metal. The extension point of the magnet, however, does not enter the vitreous.

Haab's Operation.—This may be performed as follows, according to this distinguished operator's directions:

After the usual aseptic preparations and thorough cocain anesthesia of the eye, the operator assumes one of the two positions shown in the accompanying figures.

[1] Canadian Medical Quarterly, May, 1919.

In the majority of cases—that is, in all those in which a small- to a medium-sized splinter is probably present—the center of the cornea should be placed exactly



FIG. 375.—Showing the use of the large magnet in extracting an iron spicule from the eye (Haab).

opposite the pole of the magnet. If the presence of a large splinter is suspected the pole of the magnet should first be allowed to act at some distance from the eye. The patient is told to look in the direction of the pole of the magnet. The first



FIG. 376.—Showing the use of the large magnet in extracting an iron spicule from the eye (Haab).

closure of the current may bring the foreign body behind the iris. If it does not, the current must be repeatedly opened and closed. If now there is no bulging of the iris, more lateral portions of the cornea are successively brought opposite the

pole, but the region of the ciliary body must be scrupulously avoided. To draw the splinter forward, from behind the iris, through the pupil into the anterior chamber is not always an easy matter, although, if it is smooth, it usually comes without difficulty. Occasionally iridectomy is necessary, although Haab has not found this requisite in his personal experience. According to Lang's suggestion, a smooth steel spatula, attached to the magnet, may be carried through a corneal incision behind the iris where the splinter is lodged. In uncomplicated cases after the splinter reaches the anterior chamber it may be removed through a suitable corneal incision by introducing the extension point of a small magnet, although Haab himself finishes the operation with a large magnet. Each case must be carefully considered and the technic varied according to the conditions.

The *anterior route* was the operation of choice among British surgeons during the war, and is of many of our own surgeons in army and civilian practice. The technic is closely similar to the one just described. The magnet's extension point is brought in contact with the pole of the cornea, the current alternately turned on and off, in order to draw the body through the suspensory ligament of the lens into the posterior aqueous chamber. When the iris is seen to bulge, indicating the presence of the body behind it, the current is turned off and the direction of the eye so changed that the full force of the magnet when the current is again turned on shall be parallel to the surface of the lens. The body having been drawn gradually into the anterior chamber, a small opening is made with a narrow keratome about 3 mm. below the limbus, and no aqueous spilled if possible. The extension point of the magnet is directed next outside of the cornea over the foreign body, which is coaxed along the posterior surface of the cornea into the corneal incision, through which it is drawn with the aid of a small hand magnet.

If the *scleral route* is chosen, at the point of election, as determined by *x*-ray localization, after raising a suitable conjunctival flap, a small meridianally placed incision is made through the sclera, to which the magnet is applied, and through which, while it is retracted with small, non-magnetic retractors, the body is withdrawn. The conjunctival flap is next replaced and sutured into position.

In the presence of a conspicuous wound of entrance indicating a large foreign body, its removal along the track of the wound is the usual procedure. The treatment of eyes from which foreign bodies have been extracted is the same as that which has been recommended for scleral wounds (pages 326–332).

Extirpation of the Whole Contents of the Orbit (*Exenteration*).—This is the operation necessary in certain cases of malignant disease.

The eyeball having been removed in the ordinary way, an incision is made through the outer commissure to the edge of the orbit. The lids having been widely separated, the tissues back of them and the periosteum within the orbital margin are divided with a scalpel. Next, the periosteum is separated to the apex of the orbital cavity, where the entire mass of tissue is detached with strong curved scissors or other suitable instrument. Bleeding, which is sometimes considerable, may be checked by packing with the surgical gauze or pressing surgical wax against the bleeding area or, if necessary, by the actual cautery. The cavity is loosely packed with iodoform or ordinary sterile gauze, one strand of which may be left at the outer commissure for drainage. Next, the ciliary borders, including all hair-follicles, are removed, and the lids, thus prepared, are sutured together up to the point where the gauze strand protrudes. The packing should be removed at the end of twenty-four hours. In favorable cases the lid skin will be retracted inward and completely line the socket. Thiersch grafts are also used for this purpose. It may happen that the eyeball is so involved by the malignant disease which is present that its extirpation as the first step of the operation is not feasible. The operator then proceeds as before described, removing the eyeball with the entire mass of tissue. If the lids are not sutured in the manner described, as considerable contraction of the socket takes place, its granulating surface, as stated, may be covered

with epidermic grafts. Many attempts to adjust a large prosthesis have been made.

Removal of Tumors and Cysts from the Orbit.—Tumors in the anterior portions of the orbit may be reached by an incision similar to that already described in connection with deep-seated purulent pockets (see page 363), and the growth removed by an ordinary dissection. Occasionally, in favorable situations, such growths may be reached by a dissection through the conjunctiva.

If the growth is an angioma, and is encapsulated, it may often be removed in similar manner by a slow dissection, without much loss



FIG. 377.—Spindle-cell sarcoma of the orbit which was removed, with preservation of the functions of the eye, through an incision beneath the entire orbital arch.

of blood. If non-encapsulated, and especially if it protrudes and involves the skin of the lid and brow, it is a much more difficult procedure. To a certain extent the hemorrhage can be controlled, as Knapp suggested, by pushing a horn spatula beneath the upper lid, between the eyeball and the orbit, which may be manipulated to act as a controller of hemorrhage, while the dissection proceeds from the skin surface. Although the main body of the angioma may thus be removed, it is often impossible, without sacrifice of too much tissue, to extirpate those portions of it which involve the skin of the eyelids and eyebrow. These, however, may disappear later, or may be treated by electrolysis. Recent investigations indicate that they may be successfully treated by applications of liquid air.

Sometimes encapsulated sarcomas, endotheliomas, and certain non-malignant growths, especially in the anterior portion of the orbit, may be reached without sacrificing the eyeball, according to a method advocated by Lagrange and H. Knapp, namely, first severing, if, for example, the growth is on the inner side, the internal and perhaps the inferior rectus, which are secured by threads, next separating the conjunctiva, and gradually dissecting out the growth through the opening thus made. After controlling the hemorrhage the severed recti muscles are sewed in place exactly as in the operation of advancement.



FIG. 378.—Result after removal of sarcoma of orbit. (See Fig. 377).

Again, the growth may be reached by means of a dissection which begins with an incision which extends beneath the entire length of the orbital arch and is continued slightly downward and outward along the outer margin of the orbit. Should it become necessary to divide an ocular muscle it can first be secured in the manner just described. This operation, carefully performed, in suitable cases, replaces resection of the temporal wall of the orbit and produces much less deformity (see Figs. 377, 378). In point of fact, except where the growth is within the muscle cone, in which circumstances Krönlein's operation or one of its modifications is indicated, almost always tumors and cysts can be satisfactorily reached in the manner described. Orbital cysts, dermoids, serous or blood cysts are treated in the same manner as

growths, the dissection proceeding either through an incision along the orbital margin or, if conditions are favorable, through the conjunctiva, great care being taken to remove every particle of the cyst wall, often a difficult procedure. In some instances the cyst elaboration is so extensive that the eyeball cannot be saved.

Exostoses and **osteomas** growing from the wall of the orbit, or pushing their way into it from the ethmoidal or frontal sinus, may be reached by an ordinary dissection through an incision along the orbital margin, with the usual precautions to avoid the pulley of the superior oblique, the tendon of the levator, and the lacrimal gland. After the body of the growth is fully exposed, it may be chiseled from its position in the ordinary manner or, if it is very dense and resisting, its base may be perforated several times with a drill suitably attached to a dental



FIG. 379.—Psammosarcoma of orbit. (See page 668.)

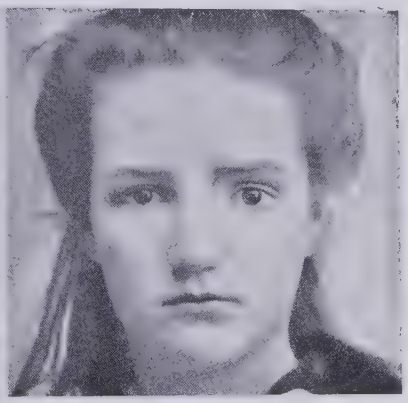


FIG. 380.—Psammosarcoma of orbit. Result after operation; eye functions normal.

engine. It is next broken from its position with a stout pair of forceps, all rough spicules of bone carefully smoothed away, and the wound closed. (See also page 668.)

Resection of the Temporal Wall of the Orbit (*Krönlein's Operation*).—The operation, following Haab's directions, begins by dividing the soft parts with a curved incision (Fig. 381), which should be about 7 cm. in length in adults and 4 to 5 cm. in children, which commences above the supra-orbital margin and describes a gentle curve along the outer edge of the orbit to the upper edge of the zygoma, where it is bent backward and ends at the center of this structure. The center of this curved incision should bisect a horizontal line which connects the outer canthus with the outer orbital margin, and here should be sufficiently deep to expose the opening of the orbit, while above and below only the skin and fascia and muscular layer are at first divided. Next, at a position corresponding to the central portion of this incision, a strong elevator is introduced, with which the periosteum is separated from the external orbital wall. The inferior orbital fissure is now localized, and, beginning at the anterior end of this fissure, the bony wall of the orbit is cut through with a chisel or with an electric saw, up and out to a point a little above the external angular process of the frontal bone, the line of incision being, for all

practical purposes, along the suture between the great wing of the sphenoid and the malar bone, and outward and forward over the external surface of the malar bone in a line above the insertion of the zygomatic arch. Thus, a wedge-shaped piece of bone is formed, and with its muscular and cutaneous attachments is forced backward, giving free access to the orbit, which will be still partly covered with the periosteum. The latter must now be split from before backward and separated with retractors. This brings into view the external rectus muscle, and, if necessary, this may be divided near its tendinous insertion after the introduction of sutures, with which later the divided ends are united, or sometimes the muscle may be pushed aside and the dissection continued to the apex of the orbit. With suitable retractors the orbital fat and ocular globe are pushed aside. After the exploration is complete, and this must sometimes be carried to the nasal side and the growth removed, the osteoplastic flap is replaced, the periosteum stitched with fine catgut sutures and the soft parts with silk. The question of drainage must be decided by the conditions remaining after operation. The usual full antiseptic dressing is applied, great care being taken that the lids cover the cornea, especially if the latter structure is anesthetic.

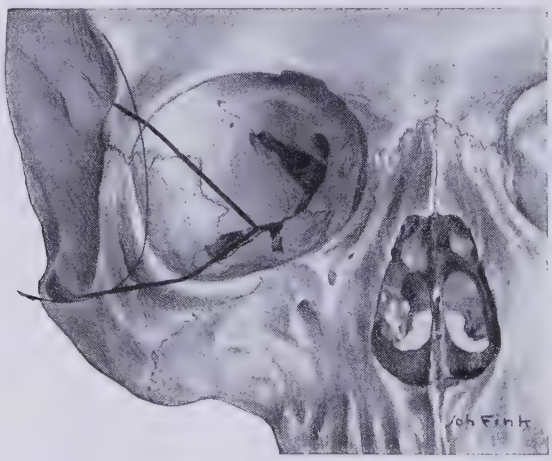


Fig. 381.—Skin incision (curved line) and bone incisions (heavy lines) in Krönlein's operation (Haab).

Various complications have occurred after this operation; for example, outward limitation of the eye owing to injury to the abducens, ptosis, sinking of the eyeball, and infection.

Indications.—Domela, Haab, and other writers have classified the indications for Krönlein's operation as follows: Retrobulbar cysts; tumors of the optic nerve and its sheath; neurofibroma of orbit (Parker); retrobulbar vascular growths, for example, cavernous angioma, lymphangioma, aneurysms, and varicose dilatations of the orbital veins; deep-seated foreign bodies in the orbit and exploration of the orbit in doubtful cases in order to establish a diagnosis. The operation has also been performed in deep orbital abscess, to open the sheath of the optic nerve in choked disk, and even for the removal of subretinal effusions (Müller). The two last indications are of doubtful value; certainly in choked disk a far better operation is a decompression-trephining.

Gifford recommends as a substitute for the Krönlein operation a definite resection of the outer wall of the orbit. He makes a horizontal incision $2\frac{1}{2}$ to 3 inches long, beginning $\frac{1}{4}$ inch from the outer commissure, care being taken not to open into the conjunctival sac. Next, the lips of the wound should be widely separated, and the periosteum pushed back from the outer side of the bone. Following this, with strong bone forceps the outer margin of the orbit and as much of the outer wall as desired is removed. Finally, the periosteum of the orbit is opened, and the operation terminates in the usual manner.

OPERATIONS FOR CATARACT

The following methods constitute the most important varieties of operation which are practised for the cure of cataract:

Extraction without iridectomy, so-called simple extraction; extraction with iridectomy, so-called combined extraction; extraction in the capsule, with or without iridectomy; extraction after subluxation of the lens with capsule forceps; linear extraction; the needle operation, or discission; and removal of lens by suction (phaco-eresis). The old operation of *reclination*, *depressing*, or *couching*, as it has been variously called, by which the lens was forcibly thrust down into the vitreous, is rarely practised at the present time, although some surgeons have suggested that the operation is advisable in patients greatly enfeebled by age or other infirmities, if chronic conjunctivitis or dacryocystitis fails to yield to treatment, in lunatics, imbeciles, and others whose actions cannot be controlled, and particularly if one eye has been lost by intra-ocular hemorrhage.

1. **Needle Operation** (*Discission—Operation for Solution*).—In this operation the capsule of the lens is opened, the aqueous humor ad-



FIG. 382.—Bowman's stop needle.



FIG. 383.—Knife-needle.

mitted to the lens matter, and absorption thus promoted. It is applicable to congenital and juvenile cataracts, and to some traumatic cataracts, and is rarely employed after the fifteenth year.

The instruments required are two cataract needles (lance-headed or knife-needle, according to the fancy of the operator), a stop speculum, and fixation forceps. The eye in this and all operations of similar character should be prepared in the manner described on page 761.

After the induction of general anesthesia in young children, or the use of cocain in older subjects, and full dilatation of the pupil, the operation is thus performed:

The lids being separated by the stop speculum, the surgeon fixes the eye with forceps, and enters the cataract needle through the cornea at its outer margin or at the limbus and carries it across to the center of the pupil, where the point is turned to the lens, and a laceration made in the capsule by depressing the handle of the instrument with a lever-like movement. Two cuts are made at right angles to

each other, and the lens matter may then be slightly broken up with the point of the needle. Care must be taken not to use so much force as to dislocate the lens, and not to lacerate too freely in the first operation, lest the lens substance, swelling up from contact with the aqueous humor, should produce injurious pressure on the iris and ciliary body. The operation usually has to be repeated at intervals, the second operation being performed after the swollen lens matter caused by the first incision has disappeared by absorption and the eye has become perfectly quiet.

At the second operation the needle may be used more freely, or two needles may be used in the manner shown in Fig. 384. The points enter the lens substance and the handles are approximated, thus making a decided separation in the remaining opaque matter. In order to prevent too deep entrance of the needle it is sometimes constructed with a shoulder (stop needle; see Fig. 382).

Instead of repeating the needlings, the first discission may be very free, with the understanding that in a few days it shall be followed by a *linear extraction* and a few

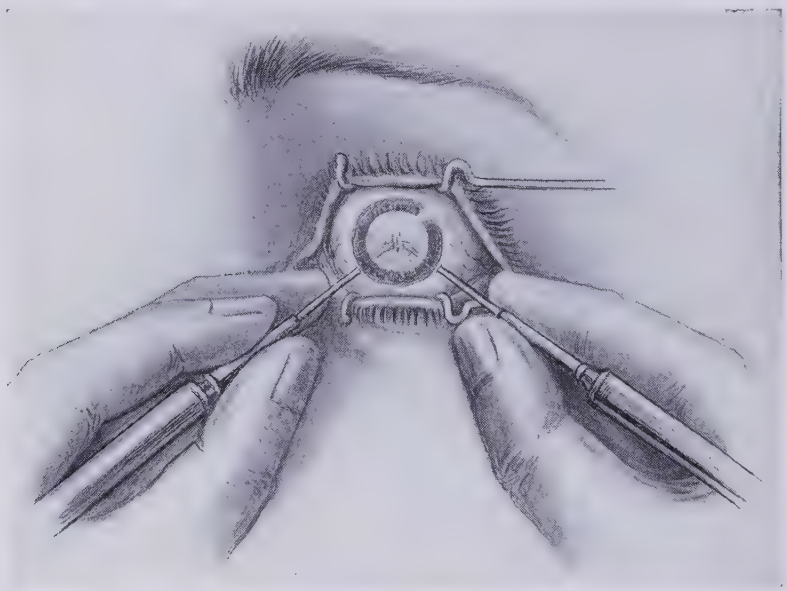


Fig. 384.—Discission with two needles.

weeks later by division of the capsule (C. F. Clark). This procedure obviates the delay, often tedious, in obtaining absorption of the lens by successive discission.

Ziegler's Method.—In congenital cataracts, in place of repeated discissions as described, or free discission followed by linear extraction, Ziegler recommends a complete discission by the V-shaped technic as this is described and illustrated on page 731; the knife needle must pass entirely through the lens into the vitreous. In spite of the extensive laceration of the lens tissue secondary glaucoma is said not to develop; the pressure in the iris angle is relieved because the posterior capsule has been opened. In Ziegler's experience only one operation is required to effect complete absorption of the lens.

After-treatment.—The conjunctival sac should be irrigated with boric acid or physiologic salt solution, atropin freely instilled, and pupillary dilatation maintained during the entire treatment. Both eyes should be lightly bandaged.

Decided reaction, with hyperemia of the iris, pain, and ciliary

congestion, indicates a more frequent use of atropin, dionin, and iced compresses. Great swelling of the lens matter, in addition to the symptoms of iritis, may give rise to a glaucomatous state. In these circumstances the mydriatic must be discontinued and the lens matter which has escaped into the anterior chamber must be evacuated by a *linear extraction* or, what is practically the same thing, by a free paracentesis of the cornea. The *suction method* may also be employed in these circumstances, but irrigation of the anterior chamber is more satisfactory.

For the *removal of the lens in high myopia* (see page 146) needling is employed. According to W. E. Lambert, the Fukala method, that is, needling the lens followed by a linear extraction, yields the best results in young subjects. If the patient is fifty or more years of age, and the lens is more or less cataractous, Lambert advises a preliminary iridectomy with subsequent extraction of the lens in the usual manner.

2. **The Suction Method for Soft Cataract.**—This operation is specially adapted for completely soft or fluid cataracts, and is also used, as has been stated, to remove lens matter which has been broken up by discission or by traumatism. It is performed as follows:

The pupil being dilated with atropin, the anterior capsule of the lens is freely lacerated with two needles. A small wound is made with a keratome passed obliquely through the cornea between its center and periphery. Through this opening and into the lens matter the "suction curet" is passed. This consists of a curet roofed in to within 2 mm. of its extremity, with a handle and a piece of India-rubber tubing furnished with a mouth-piece, which the operator applies to his lips and gently sucks out the lens matter into the syringe. This is Teale's method.

The same may be accomplished by using the syringe of Bowman, in which a sliding piston is worked by the hand. The point of the syringe must not penetrate too deeply, must be behind the lens matter which is to be removed, and must not be pushed back of the iris.

The after-treatment consists of rest, bandage, and the local use of atropin.

3. **Linear Extraction.**—This operation is designed for the removal of soft cataracts in persons under the age of thirty-five (or even older [Wilder]), and is employed to remove lens matter after discission. Any lens the substance of which is liquid enough to pass through a small corneal wound may be removed by this method. For traumatic cataract in patients not above forty years of age it should be the operation of choice. A preliminary discission is of advantage. Wilder recommends the operation, according to a special technic (Agnew's method), in membranous cataracts.

The following instruments are necessary: A keratome or lance-shaped knife, fixation forceps, cystotome, curet, and stop speculum. The operation is performed as follows:

The surgeon fixates the eye with forceps, after the introduction of the stop speculum, wide dilatation of the pupil having previously been obtained, introduces the keratome about 1 mm. within the margin of the cornea, or just at the anterior edge of the corneoscleral margin (Wilder), and makes a wound 5 mm. wide. The

instrument is now carefully withdrawn, with a slight lateral motion to increase the size of the wound if necessary, and a sharp cystotome is introduced and the capsule of the lens is freely lacerated. The soft lens matter is next made to extrude by counterpressure on the cornea with a metal spud, the outer lip of the corneal wound at the same time being depressed with a curet. This is a *simple linear extraction.*

The same manipulations may be performed, assisted by an iridectomy after the corneal section, a small segment of the iris being withdrawn either with hook or forceps and excised. Instead of using the cystotome to open the capsule of the lens, some operators employ the keratome after making the incision in the cornea by causing the instrument to dip directly into the lens, from which it is next slightly withdrawn, and as it is pressed gently backward the wound gapes and through it and over the surface of the keratome the soft lens matter exudes. Any remnants of lens material can readily be removed by irrigating the anterior chamber with physiologic salt solution.

The after-treatment consists of bandage, atropin, and rest in bed until the eye is quiet.

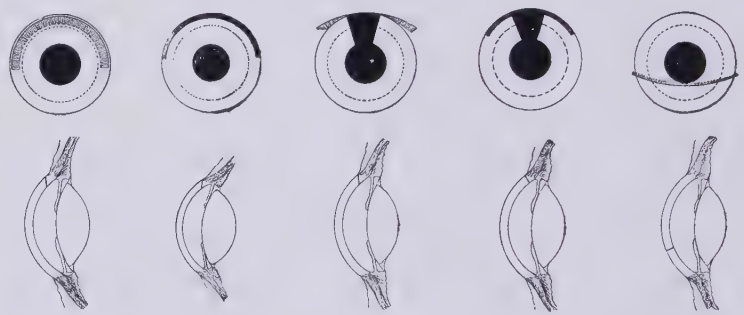


FIG. 385. FIG. 386. FIG. 387. FIG. 388 FIG. 389.

FIG. 385.—Flap extraction.
FIG. 386.—Modified flap extraction (Knapp's section).
FIG. 387.—Modified peripheral linear incision.
FIG. 388.—Short 3-mm. flap with iridectomy.
FIG. 389.—Corneal incision below.

4. **Extraction of Hard Cataract.**—It would be impracticable to indicate the numerous modifications which have been employed in this operation, than which, as the late Dr. Noyes has said, no surgical procedure has been more carefully studied and elaborated in every detail. Hence only a few well-recognized methods will be described.

(*a*) **Extraction without iridectomy, but with capsulotomy,** is often called *simple extraction.* The author is accustomed, following the directions of the late Dr. H. Knapp, to proceed as follows: The corneal section for full-sized cataracts comprises exactly the upper half of the cornea; for smaller, Morgagnian and soft cataracts, somewhat less. A perfect section passes in its whole extent exactly through the transparent margin of the cornea, the knife (see Fig. 394) remaining in the same plane throughout, particular care being taken that in completing the section the blade of the knife is not turned forward nor backward. In many cases a small central conjunctival flap is formed, which is an advantage. (For steps of operation see pages 766–768.)

(*b*) **Extraction with iridectomy and capsulotomy** is often called *combined extraction.* The peripheral linear extraction of von Graefe is no longer performed.

A useful procedure is the following: A Graefe cataract knife is entered exactly at the corneoscleral junction at the outer extremity of a horizontal line which would pass 3 or 4 mm., according to the size of the cataract, below the summit of the cornea. Counterpuncture is made at a similar point directly opposite, and a flap is cut which embraces one-fourth or one-third of the cornea. A small conjunctival flap may be made or not. (For steps of operation, see pages 764–766.)

Various *corneal incisions* have from time to time been utilized. Liebreich made an incision in the form of a curved section through the lower portion of the cornea, puncture and counterpuncture being effected in the sclera, while Lebrun caused the corneal flap to occupy the upper portion of the cornea and to be 3 mm. high, puncture and counterpuncture being made 2 mm. below the extremities of the transverse diameter of the cornea. In these operations, now rarely if ever performed, iridectomy was usually omitted.

(*c*) **Extraction without capsulotomy** is performed by many surgeons—that is to say, the lens is delivered in its capsule. The operation was formerly chiefly employed to remove overripe cataracts and cataracts complicating high myopia with vitreous changes. Pagenstecher in these circumstances expelled the lens after an incision of about one-third of the corneal circumference and an upward iridectomy. The expulsion was accomplished either by pressure or with the aid of a spoon or loop. The chief danger of the operation is the risk of extensive loss of vitreous.

Much attention has been devoted to extraction of cataract in the capsule in comparatively recent times. Three methods are: expression of the lens in the capsule after suitable corneal section by systematic external pressure (Col. Smith's method); extraction of the lens within the capsule after subluxation with capsule forceps, aided by external pressure (Knapp-Török method); removal of the lens in the capsule by suction (phaco-eresis, Barraquer's method, pages 770–772).

Preparation of the Patient and the Eye.—This should include a thorough examination of the patient, and the removal of the conditions already named (see page 462), which contra-indicate the operation.

For some days previous to the operation, as H. Knapp insisted, the eye should be protected from anything which may produce congestion, and the patient should remain in the hospital, perfectly resting his eye and body, and frequently washing his face and the surfaces and margins of the eyelids with soap and water. This simple regimen will frequently change a congested and irritated conjunctiva into a pale and shining membrane. If there is any abnormal conjunctival discharge the instillation of a solution of argyrol (25 per cent.) is recommended by some surgeons, who also employ this drug in subsequent dressings of the operated eye (Callan). Bacteriologic examination should always

be made, and if pathogenic organisms are present the operation should be postponed until suitable treatment has eliminated the source of danger. Axenfeld recommends an injection of antipneumococcic serum prior to the performance of an operation in the presence of conjunctival pneumococci. During these days scrupulous attention should be given to the nasopharynx, the tonsils, teeth, and alimentary canal. The urine should be carefully examined and the blood-pressure tested. The author, following a suggestion of J. A. Lippincott, is accustomed to spray the nasopharynx three times daily with a solution of permanganate of potassium (1 : 5000), with gratifying results. Dr. J. A. White recommends that the conjunctival sac shall be filled with bichlorid-vaselin (1 : 3000) on the night prior to the operation, where it remains until the next day.

The preparation of the skin of the region of operation, and particularly the ciliary margins, has been described on page 684. These preparations should be made at least two hours before the operation, and the eyes should be covered with squares of lint soaked in a solution of bichlorid of mercury (1 : 5000), held in place with a gauze roller. Just preceding the operation, the preparatory bandage having been removed, the ciliary margins may again be washed with soap and water, followed by bichlorid of mercury (1 : 5000), with the same precautions previously described (see page 684). Next, the conjunctival culdesac should be flushed with a tepid solution of boric acid applied with some force, or with a sterile physiologic salt solution. During these irrigations pressure should be made over the lacrimal sac in order to be sure that no deleterious secretion is contained within it. The canaliculi and lacrimal canal may be irrigated with a boric acid or saline solution introduced by means of an Anel syringe. The lids are next everted, the tarsal conjunctiva and the region of the inner canthus wiped with a pledget of cotton moistened in the boric-acid solution. The cornea should be anesthetized with three instillations of a sterile 4 per cent. solution of cocain, applied at intervals of five minutes, and the eye carefully closed and covered with the antiseptic pad after each instillation. In place of cocain some surgeons prefer holocain in 2 per cent. solution or a mixture of holocain and cocain. Just before the knife is entered the surface of the cornea should be carefully wiped with a pledget of cotton soaked in boric-acid solution. This same method of preparing an eye should be practised not only in cataract extraction and discission, but also prior to all operations requiring corneal incision—for example, iridectomy, iridotomy, etc. The author has not deemed it wise to flush the conjunctival sac vigorously with strong solutions (1 : 2000 or 3000) of bichlorid of mercury, but this practice is commended by surgeons of great experience, for example, Col. Henry Smith. Placing a preparatory dressing over the eyes during the night preceding the operation the author is satisfied is an unwise procedure. Dr. G. H. Bell advises the instillation of 2 drops of a 1 per cent. solution of nitrate of silver, which produces a marked irritation of the conjunctiva, and which is followed later by irrigation

with physiologic salt solution, prior to all operations on the eyeball. He insists on the elimination of areas of focal infection in the teeth, tonsils, and intestines. To render the lids flaccid and prevent the danger of "squeezing," the lid area may be injected with a 1 or 2 per cent. solution of procain (novocain).

Position of the Patient.—The patient during the operation should lie, according to the custom of the operator, upon an operating chair or table. If, as is sometimes advisable, the operation is performed while the patient reclines in bed, the head should rest on a moderately hard cushion or pillow, covered with a sterile sheet, another pillow at the same time supporting the shoulders, so that the position is as little strained as possible. In all circumstances the face must be turned so that uniform daylight falls upon it, or the area of operation must be illuminated by a suitable electric lamp.



FIG. 390.—Lid elevator.



FIG. 391.—Metal spoon.



FIG. 392.—Wire loop.



FIG. 393.—Cystitome.



FIG. 394.—Cataract knife.



FIG. 395.—Capsule forceps.

Instruments, Solutions, and Dressings.—The instruments required are the following: A stop speculum, a lid elevator, a large strabismus hook, a spatula, a wire loop, a spoon, an olive-tipped probe, a curet, a cystitome, capsule forceps, a pair of scissors, iris forceps, iris scissors, and the cataract knife.

The following lotions and dressings should be at hand: Atropin drops, 4 grains (0.26 gm.) to the ounce (30 c.c.); eserin drops, $\frac{1}{2}$ grain (0.0324 gm.) to the ounce (30 c.c.); cocain solution (4 per cent.); saturated solution of boric acid; two solutions of bichlorid of mercury

(1 : 5000 and 10,000), boiled distilled water containing 0.5 per cent. of chlorid of sodium and adrenalin (1 : 1000). Suitable bulb syringes and an irrigating apparatus, for example, Lippincott's, or the one described on page 464, should be ready.

For the purpose of dressings the following may be needed: Several rollers, 2 inches wide and 5 yards long, made of sterilized gauze, and sterilized oval pads of lint and absorbent cotton. A useful bandage is composed of a broad band of knitted material which is tied with four tapes, which pass above and below each ear.

Everything being in readiness, the operation may be performed as follows:

The surgeon, if he is ambidextrous, may stand behind the patient, no matter which eye is to be operated upon; if he is not, he should take his position for the right eye only, standing at the patient's side and in front for an operation on the left eye. Again, if the surgeon is ambidextrous, he may stand in front and at the patient's right side for an operation upon the right eye, and at the patient's left side and in front for an operation on the left eye.

The speculum having been inserted, the surgeon steadies the eyeball and draws it downward with the fixation forceps (it is supposed that the section is being made upward) by taking firm hold of a fold of conjunctiva below the inferior border of the cornea (some surgeons prefer fixation at the inner side, over the internal rectus tendon), enters a Graefe cataract knife exactly at the corneoscleral junction, as before described, at the outer extremity of a horizontal line which would pass 3 or 4 mm., according to the size of the cataract, below the summit of the cornea, passes it across the anterior chamber to a corresponding point upon the opposite side, and makes the counterpuncture. The knife is pushed steadily onward as far as possible, with an upward tendency, and the incision is completed by a free cutting, not a sawing or dragging movement, keeping the knife in the same plane throughout, and not turning its edge at the completion of the section either forward or backward. This maneuver will create a small conjunctival flap. If this is not desired, when the summit of the cornea is reached the knife must be turned a little forward before the completion of the flap. It is the practice of some surgeons to remove the speculum as soon as the section is completed; other operators prefer not to use a speculum, but to separate the lids with their fingers or with a lid elevator or a Smith's or Fisher's hook held by an assistant. This completes the *first stage* (Fig. 396).

In the *second stage*, or the stage of iridectomy, the fixation forceps are intrusted to the assistant (trained to hand the instruments in their proper order), who gently draws the eyeball downward, while the operator takes in his left hand the iris forceps and in his right the iris scissors. If the iris is already protruding in the wound, a small portion of it may be seized and snipped off with a single cut close to the border of the cornea. If not, the blades of the instrument must be introduced in the manner described under Iridectomy, and the iris seized, the tissue drawn out and cut off close to the cornea. It is not advantageous to make a large coloboma. If the patient is to be trusted, it is not necessary that the assistant shall draw the eyeball downward while iridectomy is being performed. The patient may simply be directed to look downward while the surgeon proceeds to remove a small portion of the iris in the manner already described. The pillars of the coloboma should now be carefully smoothed out with a delicate spatula. This completes the *second stage* (see Fig. 353).

In the *third stage*, or the stage of capsulotomy, the operator takes in one hand the fixation forceps and gently steadies the eyeball, while with the other he introduces the cystitome, held flatwise during its insertion, passes it to the bottom of the coloboma, and then turns its cutting edge toward the capsule. From this point a vertical incision is traced until the upper portion of the coloboma is reached, where a transverse cut is made. Great care should be taken to cut, and not to tear,

and the whole maneuver should be accomplished without undue pressure lest the lens be dislocated. Other methods of opening the capsule are the following: Two cuts inclined to each other are made like the limbs of the inverted letter ∀, together with a transverse cut at the periphery; or, as was recommended by H. Knapp, the capsule may be opened in its extreme periphery, with the understanding that later on the necessity for the operation for after-cataract will arise. In withdrawing the cystitome the operator should again turn it flatwise, and be careful not to drag any tags of capsule into the wound. The cystitome (Fig. 393) often employed is not a satisfactory instrument; one with a small oval knife-like blade, such as Ziegler has devised, is a much better instrument in that it easily cuts the capsule.

Many surgeons disregard the cystitome and open the capsule with *capsule forceps;* after introduction, the blades are slightly expanded and the central portion of the capsule seized in their grasp and removed. This procedure usually obviates the necessity of a secondary operation, and is decidedly preferable to the use of the cystitome. This completes the *third stage.*

In the *fourth stage,* or that of delivery of the cataract, the operator draws the eye slightly downward, or, if he has a docile patient, causes him to look downward, while the assistant raises the speculum so that its blades shall not press upon the

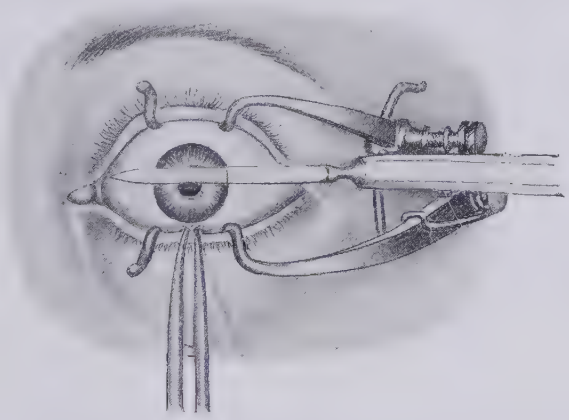


Fig. 396.—The incision in cataract extraction. Puncture and counterpuncture have been made. The section will pass in its whole extent exactly through the transparent margin of the cornea, the knife remaining in the same plane throughout.

eyeball and yet shall hold the lids away from the eye or, the speculum having been removed, lifts the upper lid with a Smith hook or with an elevator, at the same time drawing away, with his thumb, the lower lid from contact with the globe. The back of a curet or the convex surface of the metal spoon is now laid against the inferior portion of the cornea, and firm but at the same time gentle pressure is made, causing the upper margin of the lens to appear in the wound. The pressure is exercised with an upward motion to coax out the cataract, but is relaxed as soon as the major portion has been expelled, in order that no undue tension be put upon the zonula. As the cataract slips through the wound the spoon is made to follow it, catch it, and lift it out with a little sweeping motion which may at the same time remove any small fragments of the cortex which have broken off and lie at the margins of the incision. This completes the *fourth stage* (Fig. 397).

In the *fifth stage,* or that which is now called the "toilet of the wound," after the eye has been allowed to remain closed for a few moments the operator cautiously inspects the wound, after raising the upper lid with his fingers, or preferably with a Smith hook, while the patient looks downward. In this inspection he should ascertain whether the pupil is clear or whether any cortical remnants are present or tags of capsule lie between the lips of the incision. If cortical matter remains, it should

be removed as follows: The eye being turned downward, the operator makes a gentle rubbing movement in an upward direction on the cornea with the convex surface of a horn spoon, great care being taken not to press too hard lest vitreous escape. By rubbing gently in a circular manner the cortical particles will gather in the upper part of the wound, and then, while the slight pressure continues, the lips of the wound may be gently separated with the metal spatula and the expulsion of the cortical remnants effected. Blood-clot, the result of hemorrhage from the iris, may be expelled in like manner. While these manipulations are being made, the author is accustomed to flood the surface of the eye and lips of the wound with a physiologic salt solution. After they are completed, a final inspection is made, and in order to be sure that no tag of capsule remains in the wound, or that no portion of the conjunctival flap has been caught between its lips, the olive-pointed probe is gently passed from one end of the incision to the other.

Some surgeons advocate *irrigation of the anterior chamber*, which, as has already been stated, is used also in the operation of unripe cataract. In this maneuver the tip of a specially devised syringe is introduced between the lips of the wound, and the irrigating liquid injected, which causes blood-clot or cortical matter to be washed out. If irrigation is employed, two cautions are necessary: (*a*) No strong antiseptic solution should be used, certainly never bichlorid of mercury, which is liable

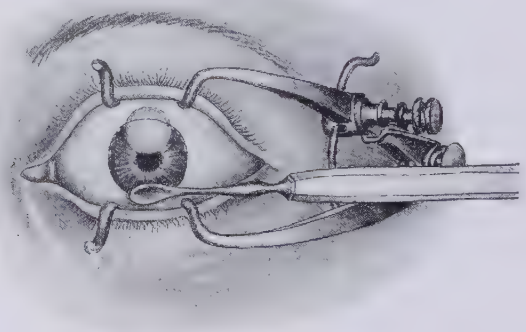


Fig. 397.—The delivery of the lens; the lens is presenting in the wound (capsulotomy has been performed).

to produce indelible opacity of the cornea. The irrigating fluid should be boiled distilled water containing 0.5 per cent. of the chlorid of sodium. (*b*) In passing the liquid from the syringe into the anterior chamber, the direction of the flow should be over the wound from within outward, and not the reverse, lest particles of blood and cortex be driven inward.

A general inspection of the conjunctival sac may now be made; sometimes a little blood-clot or a cilium may be present. In wiping away any clots, delicate pieces of sterilized gauze are suitable, or the clots may be picked up with the iris forceps. If all these manipulations have been successfully performed, the conjunctival culdesac will be free from foreign matters, the edges of the wound nicely coapted, the pillars of the coloboma as straight as possible, and the angles not caught in the margins of the wound, the pupil black, and the patient readily able to count fingers. This completes the *fifth stage.*

Extraction without iridectomy, following the method of the late Dr. H. Knapp, may be performed as follows:

After making the section according to the method already described (see page 764, also Fig. 396), the expulsion of the lens is effected by pressing the lower part of the cornea with a Daviel spoon directly toward the center of the globe. When the

lens presents in the gaping section, its exit is aided by slight strokes with the spoon on the outer surface of the cornea. If the sphincter proves to be rigid, it may be drawn backward with a wire loop or with a special iris retractor, and usually it is safer to remove both fixation forceps and speculum immediately after the corneal section and during the process of expelling the lens, or the speculum may be raised in the manner already described. If desirable, the upper lid may be elevated in these circumstances with a large strabismus hook or with a lid elevator. The pupillary space should be cleared by pressing on the cornea with the edge of the lower lid—care being taken that it does not come in contact with the lips of the wound—or, better, with the convex surface of a polished spoon. The cortical remnants are wiped away with a probe-pointed curet. During this operation the lips of the wound may be flooded with a sterilized salt solution.

The concluding steps of the operation are described in Knapp's own words: "The conjunctival flap is smoothed out by introducing the end of a polished grooved spatula, previously sterilized, into the anterior chamber, and passing it through the wound from one end to the other, stroking from within outward, in order to remove particles of lens, redress a curved-in flap, and carefully adjust the edges of the wound. This is, however, not done before the iris has spontaneously or artificially recovered its natural position. Should the corneal section be too peripheric, the best thing is to make a small iridectomy at once, for peripheric (Graefe's) sections commonly lead to large and harmful prolapses. If the iris does not spontaneously resume its position, frequently it does so when the lower part of the cornea is pressed upon with the edge of the lid. This paradoxic phenomenon may thus be explained: The iris being pinched in the tightly closing wound, pressure on the part of the cornea raises the flap and disengages the iris, which then, by its natural elasticity and contraction of the sphincter pupillæ, can resume its natural position. If this procedure fails, the iris should be pushed back with a spatula into the anterior chamber. When the periphery of the iris remains folded in the sinus of the anterior chamber, it is smoothed out with the olive-tipped point of a probe introduced into the iris angle behind the opaque corneal margin."

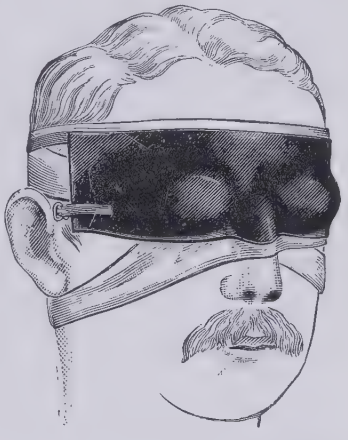


Fig. 398.—Ring's ocular mask.

The final stage of all cataract operations is the application of the dressing. Some operators simply close the lids with a strip of isinglass plaster, while others place upon them an elaborate bandage.

The author is accustomed to use the following dressing: An oval piece of soft lint soaked in a solution of boric acid is laid upon each closed lid, the margins of which have previously been liberally smeared with bichlorid-vaselin (White's ointment, see page 762); over this is placed a similarly shaped piece of sterilized cotton large enough to be flush with the eyebrow and lower margin of the orbit, and is held in place with three narrow strips of surgeon's isinglass plaster, passed from the inferior edge of the orbit to a point above the brow. Over this a very light, single piece of knitted bandage is tied by means of four tapes which pass above and below the ears. The entire dressing is usually covered with the mask devised by Dr. Frank Ring, of New York (Fig. 398). The patient is put to bed in a comfortable position in a slightly darkened room, although with the aid of the mask the latter precaution is unnecessary, and the patient may remain in the open ward of the hospital or in an ordinary room without danger. If the caruncles are tumid, or if there has been any suspicious secretion from the lacrimal sac, the author is accustomed to fill the inner canthus with dry sterile iodoform powder, which forms a

small cake and prevents access of infection to the wound. Some surgeons, for example, H. Bruns, in hospital practice, at least, pass two or three strips of plaster over the closed lids, put on top of this cotton which is held in place with strips of gauze and collodion, and over the whole a wire cage is adjusted. The patient is allowed to go home and comes daily for observation and dressing.

After-treatment.—For the first few hours, the effects of the cocain having passed away, there are some smarting and burning, but severe pain should not occur. At the end of twenty-four hours the dressing may be removed and the lids inspected. A little staining of the strip of lint is of no consequence, and if the eyelids are not swollen and there is no discharge and the delicate veins in the skin of the lids are not distended, the eyelids need not be opened and the dressing may be reapplied; or the lower lid may be gently drawn downward so as to permit the escape of tears which may have accumulated in the conjunctival culdesac, or to liberate the eyelashes if they have become inverted. At the end of forty-eight or seventy-two hours the wound may be inspected by candle-light, a drop of sterile atropin solution instilled, and each succeeding day the usual dressing reapplied; at the end of three days the dressing may be removed from the unoperated eye, and at the end of a week or even earlier the patient needs only a shade and dark glasses. Although some operators do not require cataract patients to go to bed at all, it seems to the author that it is safer to keep them in bed for two or three days. The recumbent posture too long maintained may lead to hypostatic congestion of the lungs. Sometimes elderly patients are very uncomfortable when confined to bed and become slightly delirious; in these circumstances they may be allowed to rest in an easy chair. For a few days liquid food, or at least food which does not require much chewing, should be given; after this the ordinary diet suited to the patient is permissible. For the first night or two after the operation there is no objection to the use of a mild hypnotic—barbital (veronal), 5–10 grains at bedtime, or luminal, 3 grains.

Some surgeons prefer the "open method" of managing eyes after cataract extraction—*i. e.*, no bandage is applied, but the eye is protected by spectacles made of wire gauze or similar material. With this procedure the author has no experience.

If the operation has been an extraction without iridectomy, it is proper to inspect the eye at the first dressing, usually at the end of twenty-four hours, in order to ascertain whether there has been any prolapse of the iris. Should this accident have occurred, the treatment must be pursued according to the directions given elsewhere (page 777). If the iris is in place and the pupil circular, although it is proper to change the dressings once in twenty-four hours, it is unnecessary to inspect the line of incision. All that is required is to draw down the lower lid and permit the escape of accumulated tears. As soon as the wound is closed, a drop of a sterile atropin solution may be instilled and this instillation repeated at subsequent dressings.

Accidents.—The following accidents may occur during the performance of a cataract extraction:

1. The knife may be introduced with the cutting edge turned in the wrong direction. If this somewhat inexcusable mistake should occur, the knife must be withdrawn and properly inserted. If this cannot be done, owing to the escape of the aqueous, postponement of the operation until the anterior chamber has refilled is necessary.

2. The conjunctiva in the neighborhood of the counterpuncture may become distended with aqueous humor. This produces an elevation resembling a bleb. The section should be completed as if the accident had not happened.

3. The iris may fall before the knife. The incision should be completed in the ordinary way. An irregular coloboma may be remedied by seizing the jagged edges with the iris forceps and trimming them with the scissors.

4. Free hemorrhage may occur if a conjunctival flap is made or in performing the iridectomy. Under pressure the bleeding will sometimes cease, and the operator should then endeavor to get rid of the blood in the manner already described. If success does not follow the maneuver, the cystitome may be introduced, even though everything is obscured by the blood, the capsule lacerated, and the lens expelled.

5. The wound may be too small. This defect should be remedied by enlarging the incision, which is best done with a small pair of probe-pointed scissors.

6. Undue pressure of the cystitome or the capsule forceps may partially or completely dislocate the lens. If the dislocation is partial, the eye should be closed and gentle pressure should be made with a bandage; the lens probably will right itself and can be delivered. If the dislocation is complete and the lens slips back into the vitreous, it must be removed by means of the scoop or wire loop or by pressure (see page 469).

7. The vitreous may escape before or after the expulsion of the lens. If before the expulsion of the lens, the operator should at once remove the cataract with the wire loop, which is gently inserted behind the lens. At the same time all pressure upon the eye must be removed. If vitreous escapes after the lens has been extracted, the wound should be cleared of protruding vitreous as gently and rapidly as possible and a bandage applied. The patient should be required to look upward, as a downward position of the eye favors the prolapse of the vitreous. Although escape of vitreous is an undesirable accident, its consequences are not always serious and good visual results may be obtained. If the escape of vitreous has been great, particularly if the vitreous is thin and there is tendency for the eyeball to collapse, a tepid sterile physiologic salt solution should be injected into the vitreous chamber until the globe assumes its proper contour, as has been recommended by J. A. Andrews and Herman Knapp.

8. Sometimes immediately at the conclusion of the section, or directly after the delivery of the lens, especially in old and feeble subjects, there is collapse of the cornea, which, instead of keeping its proper curve, looks like a wrinkled membrane. In these circumstances the anterior

chamber should be filled with physiologic salt solution, which will not only aid in making proper coaptation of the lips of the wound, but will prevent the sucking in of the conjunctival juices which might lead to infection.

9. Capsulotomy may not have been sufficient and pressure upon the inferior half of the cornea fails to cause the lens to present. In such a case the cystitome must be reintroduced and the laceration enlarged, or if the obstruction is due to the presence of a tenacious center in the capsule, this may be removed with capsule forceps. In most circumstances the use of capsule forceps is far preferable to the employment of a cystitome.

(1) **Extraction in the Capsule.—Smith's Method** (sometimes called *Jullunder* or *Indian Method*).—The operation is performed as follows:

A Graefe knife is entered at the corneoscleral junction and counterpuncture made in the corneoscleral junction of the opposite side, so that the incision when completed shall include half or nearly half of the circumference of the cornea. An iridectomy may or may not be done, but it is advisable, for the beginner at least, to make this part of the operative procedure.

The speculum being removed, the eyelid and brow are held away from the eye by means of a large blunt hook and the lower lid by the assistant's fingers. The patient is required to look steadily upward. In the case of an *immature lens* or of a *hard cataract* the operator next presses back with the point of a strabismus hook toward the optic nerve, the point of the hook being placed over the lower third of the cornea. This pressure must be steady, and the point of the hook should not be removed until the upper edge of the lens tilts forward. The moment the lens is seen to be dislocated the point of the hook is gradually turned more and more toward the wound, pressure during all of this time being maintained, so as to keep the lens up to the sclerotic margin, the pressure with the hook becoming lighter and lighter and the hook gradually sliding under the lens. At this stage the lens is delivered.

If the *tension of the eye is low*, the emerging lens should be followed with a spatula as well as with a hook, and sometimes, in these circumstances, a light counter-pressure with a spatula above the wound is indicated.

In the case of *intumescent lenses* and *Morgagnian cataract*, the capsules of which are extremely delicate and liable to burst, pressure with the point of the strabismus hook is made sufficiently deep over the lower border of the lens to cause it to dislocate below and turn a half somersault, the pressure and traction always being made over the zonula. As soon as the lens turns up into the wound the operator ceases to make traction toward the patient's feet and directs the pressure backward, next backward and upward, and finally more and more toward the wound, folding the cornea beneath the lens until it falls outside of the cornea. As the capsule has not yet been dislocated from the zonula in the neighborhood of the wound, the hollow of the curve of the strabismus hook should be made to sweep along between the lens and the wound to complete this detachment. It is advisable in these circumstances to keep up sufficient tension on the eyeball with the spatula placed on the cornea in order to prevent the lens from slipping back into the eye.

Hypermature cataract is difficult to dislocate, and the operator should proceed with the strabismus hook as in the case of immature and hard cataract, but a spatula in his left hand should be in position at the margin of the wound to drop behind the lens the instant its edge appears. As it is dropped almost straight down into the eye, the back of the spatula must be placed against the sclerotic margin of the wound and the lens pressed against it with the hook from the outside. The lens, therefore, slides along the inclined plane of the spatula.

Great care should be taken to return the iris to its proper position, whether or not an iridectomy has been made, and with a suitable repositor the operator should

release any part of the iris from the sclera or from the angles of the wound. If this is carefully done, the eye need not be dressed until the expiration of the eighth day, and many of the complications which have been described may be attributed to meddlesome dressings and meddlesome inspections. If vitreous escapes, it should be snipped off with the scissors in the usual manner.

According to Colonel Smith, cataracts occurring in children and young persons are not suited to this operation, because it is practically impossible to dislocate their lenses. Much difference of opinion exists as to the value of this operation and its permanent place in surgical practice. In this country it has been practised by a number of surgeons (Vail, Timberman, W. A. Fisher, A. S. Green, L. D. Green, and others).

One of the objections to the Indian operation is that the percentage of loss of vitreous is much higher than in the ordinary extractions of cataract. Colonel Smith has reported vitreous loss as high as 8 per cent., while Vail, in a series of uncomplicated cataracts, did not exceed 2 per cent. of vitreous loss, and therefore he believes, to use his own expression, that vitreous escape is not the argument to be used against Smith's technic. W. A. Fisher, who has modified the Smith method, although admitting the disadvantage of frequent vitreous loss, believes this can be overcome by careful technic and that Smith's operation should be the procedure of choice. Naturally, the ordinary accidents of cataract extraction are liable to occur, but in Smith's Indian service they were not greater than those which occur in ordinary extraction. It is the author's impression, based on a limited personal experience and upon his observation of certain operators who are familiar with the Smith technic, that while this operation may retain a place in ophthalmic surgery, especially in the extraction of unripe cataracts, it is not likely to drive from the field those procedures which have for years been firmly and favorably established.

(2) **Extraction After Subluxation of the Lens with Capsule Forceps.**—Several operations for the extraction of cataract in the capsule after subluxation of the lens with capsule forceps have been devised (Stanculeanu, A. Knapp, Török).

Arnold Knapp's Method.—"After the usual preparation and the instillation of 1 drop of atropin, under holocain anesthesia, the Koster speculum is introduced and left in place until the operation is completed, unless there is danger of or actual prolapse of vitreous. An assistant is necessary only in the presence of complications. The section must be large and should be just short of half the corneal circumference with a conjunctival flap. After the iridectomy, the capsule forceps is introduced to a point below the center of the pupil, the branches are then allowed to separate broadly, and a distinct knuckle of capsule is grasped. The grasp should not be too tight lest the capsule be torn, but sufficiently firm to exert traction on the periphery of the lens capsule. The closed branches of the forceps are gently moved from side to side, up and down or rotated, and the capsule can be seen to follow in the various directions. When the dislocation has succeeded, a part of the margin of the cataract in the capsule appears free in the pupillary space. The portion dislocated is usually below, generally slightly to one side or the other, with the upper attachment unruptured. The forceps is then released and withdrawn. Pressure is exerted straight back on the lower part of the cornea with Smith's hook, and the cataract can be seen to turn a somersault: it "tumbles," in other words, as Smith

calls it, and is delivered feet first. When the entire lens has been delivered, it will be found adherent above, where it is finally separated by a lateral stroking motion. In some cases the head presents first; the delivery is then slower, and counterpressure must be applied at the scleral margin. The iris columns are then carefully replaced. The coloboma should ultimately not appear any different from that after an ordinary extraction." Török expels the lens by intermittent external pressure combined with traction by the capsule forceps.

(3) **Extraction of Lens by Suction** (*Phaco-eresis, Barraquer's Method*).—Suggested by Hulen, and recently elaborately put into practice, according to a special technic, by Barraquer, this method has attracted much attention in recent times.

The pupil of the cocainized eye is dilated (euphthalmin or atropin) the eyelids having been injected with a 1 or 2 per cent. solution of procain (A. S. and L. D. Green recommend that 1 c.c. of same solution shall be injected deeply into the orbit), the upper lid is controlled by an assistant with a hook (Smith's hook, for example), while the lower lid is drawn downward. The corneal incision, which should include two-fifths of the cornea, is made in the usual manner in the limbus, and completed with a conjunctival flap. A small peripheral iridectomy is made. The cup of the specially devised suction apparatus (erisphake) is introduced into the anterior chamber and slid under the iris, and is applied accurately to the lens and suction made, associated with a gentle rocking movement of the handle of the instrument, and the lens withdrawn. After toilet of the wound, a drop of an oily solution of eserin may be instilled. Both eyes are bandaged. Foster Moore has designed a modified suction cataract extractor, to replace Barraquer's erisphake, and A. S. and L. D. Green have introduced alterations in the apparatus (a foot-controlled motor) and have combined the suction-traction with pressure on the lower part of the cornea to facilitate the delivery of the lens.

Cataract extraction without iridectomy may be performed according to *Chandler's method*, in which a small piece of iris, 1 mm. in diameter, is removed, making a very small, round opening as near the root of the iris as possible. This facilitates drainage and prevents iris prolapse. In Angelucci's modification of cataract extraction of fixation is on the superior rectus muscle, and the entire operation is completed without speculum or aid of assistant.

Preliminary Iridectomy.—Some operators, almost as a rule, perform a preliminary iridectomy and extract the cataract several weeks later, because by this method the dangers of the final operation are lessened. It is recommended in any case where serious complications are apprehended, where for any reason an extraction in one eye has terminated unfavorably or where the cataract is not ripe, when the intra-ocular tension is raised, and in diabetic cataracts to lessen the danger of iritis (Terson).

Preliminary Capsulotomy.—Operative procedures designed for the purpose of ripening immature cataract are described on page 464. Homer E. Smith advises preliminary capsulotomy and operates as follows:

The capsulotomy knife is thrust through the middle of the superotemporal quadrant of the right cornea and is made to reach the lowest point of the dilated pupil and to rest on the vertical meridian of the lens. The handle is now raised until the blade penetrates the capsule. Next, the handle is made to describe an arc

of a circle away from the operator, the capsule being incised along the vertical meridian, while the shank is gradually withdrawn. The point of the knife is next released, the shank introduced, the blade carried to the nasal margin of the pupil, and the capsule incised along the horizontal meridian. If the left eye is to be operated upon, the knife is entered in the temporo-inferior quadrant of the cornea and the first incision is from above downward. Eserin (gr. 1½ to f℥j—0.097 gm. to 30 c.c.) is instilled and the eye bandaged. At the expiration of six hours the lens is extracted in the usual manner. The object of this operation is to separate the capsule from the lens and bring about a union of the nucleus and of the cortex.

Smith's procedure has received the endorsement of a number of surgeons. Apparently there is no harm in extending the interval between the two operations to twenty-four hours (Hansell and Shannon).

To prevent iris and vitreous prolapse a number of operators have placed, prior to the extraction, a suture; *Kalt's corneal suture* in this respect being well and favorably known is especially recommended by E. C. Ellett. The point of a sharp needle, armed with fine linen thread, is passed into and out of the *substantia propria* on one side of the proposed section and next obliquely through the opposite border of the limbus and through the scleral tissue. The loop thus formed is drawn aside, the incision completed, and after the delivery of the lens the threads are tied in a single knot. *Sliding conjunctival flaps*, which cover the line of incision, for example, the Van Lint flap, recommended in certain cases by L. Webster Fox, have also been used to prevent iris prolapse and infection. A number of operations whereby a *subconjunctival extraction of cataract* is performed have been devised, for example, by Dimmer, and in this country by the late Dr. Frank Todd. Some operators as a rule form a *conjunctival bridge* beneath which the lens is extracted, differing, therefore, from a large conjunctival flap in its shape and because its upper end remains undivided.

Anomalies in the Healing Process After Cataract Extractions.—*Pain.*—Should pain occur and not be due to the circumstances already mentioned, but become violent in character, either in the earlier stages after the operation or some days afterward, one of three things may be apprehended: intra-ocular hemorrhage, suppuration of the wound, or iritis.

Expulsive Intra-ocular Hemorrhage.—Usually, soon after the operation has been completed, the patient complains of very severe pain, or vomiting may occur and the dressings begin to be stained with blood. On removal of the bandage a clot of blood will be found protruding through the palpebral fissure, and on raising the lid the anterior chamber is seen to be full of blood and the corneal wound gaping widely. As soon as the symptoms of this accident are manifest, the patient should be placed in an upright position and a hypodermic injection of morphin administered. The blood should be carefully removed, the conjunctival sac washed out with a bichlorid solution (1 : 8000), and a full antiseptic dressing applied. The dressings should be changed once or twice daily. In this way it may be possible to avert suppuration, even though the eye remains blind. If the hemorrhage should continue and the pain become intense, enucleation is necessary.

Anterior Chamber Hemorrhage.—Not very infrequently hemorrhage takes place in the anterior chamber (about 4.5 per cent. in Wheeler's statistics), usually soon after the extraction, but sometimes at later periods, even after one month. Such hemorrhages are often the result of slight injuries; albuminuria, glycosuria and arteriosclerosis are predisposing causes. The bleeding may be spontaneous and is sometimes recurrent. Usually the hemorrhage disappears rapidly; iced compresses should be applied, although occasionally hot compresses, especially if absorption is slow, may be useful; lactate of calcium may be administered; dionin locally is of service.

Suppuration of the Wound.—According to Treacher Collins, purulent infection is more common in old people than in young, and the tendency is greater between sixty and seventy than between seventy and eighty, though it is certainly greater between eighty and ninety than between sixty and seventy. It may be caused by lacrimal tract infection, inflammation of the upper respiratory tracts, sinus disease (other areas of focal infection have been incriminated), conjunctivitis, blepharitis, by infection introduced at the time of the operation, or during the first dressing of the eye, for example, a non-sterile atropin solution. Suppuration commences on the first, second, or third day, more rarely on or after the fifth day, but sometimes as late as the thirteenth day. Investigating the microbial causes of operative infection Morax found that the pneumococcus was most frequently the active agent. Other infections were due to steptococci and staphylococci.

The symptoms are pain, swelling of the lids, chemosis of the conjunctiva with undue secretion, haziness of the cornea, turbidity of the aqueous, and the formation of a slough along the margins of the wound.

Two terminations are possible: The suppurative process may be limited, so that at the end of the inflammation the pupil is closed and the iris drawn upward, or the entire globe may participate in a general destructive inflammation (purulent panophthalmitis).

If the suppuration is limited to the margin of the wound, prompt treatment may be of avail. The conjunctival sac should be carefully disinfected, the lips of the wound gently parted after removal of the slough and irrigated with a bichlorid solution, and the whole line of incision freely cauterized with the actual cautery or with liquid carbolic acid. In other words, the treatment is practically that which has been advised for a sloughing ulcer. At each subsequent dressing the lips of the wound should be parted with a probe and the anterior chamber drained. Argyrol solution (25 per cent.) may be instilled into the conjunctival sac, and its introduction into the anterior chamber has been advised. Hansell recommends the injection into the anterior chamber of a few drops of a solution of bichlorid of mercury (1 : 1000). Ziegler advises the constant application of ice (which is undoubtedly superior to hot compresses so often advised) and the application of formalin to the wound and its injection into the anterior chamber. Subconjunctival injections of bichlorid of mercury or cyanid of mercury have been recommended in these circumstances, and the introduction of iodoform

into the anterior chamber has been suggested, and successes have been reported. In recent times postoperative suppuration has been treated by means of *vaccines;* for example, the injection of Wright's *anti-staphylococcic vaccine*. *Antidiphtheritic serum* has been effectual in some cases (see page 282). A *bacterin* prepared from the micro-organism which is active has in more than one instance been successful, and should certainly be given full trial. The bacterin treatment may be combined with the internal administration of urotropin. Should the infecting micro-organism prove to be the pneumococcus, *ethylhydrocuprein* may be tried (see page 284).

If the infection manifests itself in the form of a *ring abscess*, treatment is usually unavailing, and the eye passes into a state of panophthalmitis and requires the treatment for that condition which has already been detailed. Suppuration, instead of beginning in the cornea, may sometimes commence in the iris and even in the vitreous, and the

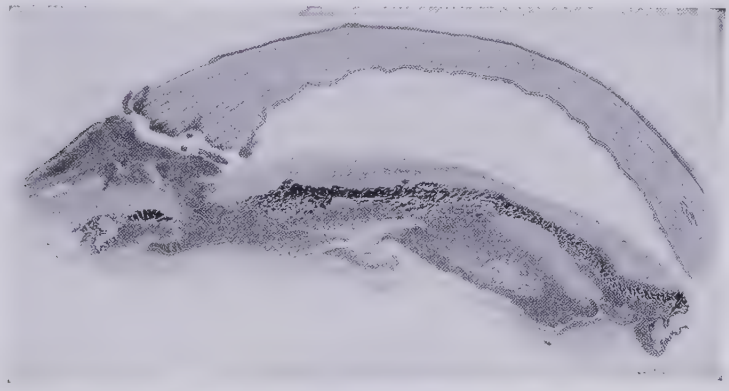


FIG. 399.—Section of an eye with postoperative infection which began on the ninth day after spontaneous reopening of the wound. Notice the dense infiltration of the wound edges and that the lens capsule has been caught in this material between the lips of the wound.

process go on to a rapid destructive panophthalmitis. An abscess of the posterior part of the vitreous has been observed as the primary focus of infection, the lips of the wound being unaffected. It is a clinical fact that if one eye has been lost on account of postoperative suppuration, the other eye, if submitted to operation, is in grave danger of meeting a similar fate.

Iritis and Iridocyclitis.—It is not uncommon for attachments to form between the capsule of the lens and the margin of the pupil or of the coloboma. These synechiæ usually are not of serious consequence. Iritis itself, with the usual symptoms of this condition, generally sets in about the fifth day, but may be delayed to the tenth day. It has been attributed to an imperfect toilet of the wound, with the retention of pieces of cortex, and sometimes to early exposure of the eye, but really should be regarded as a manifestation of infection with the staphylococcus as the predominating organism (Morax). If the ciliary body is

involved, the gravity of the situation increases and the process may terminate in distortion and closure of the pupil, with exuded lymph. Iridocyclitis may last for weeks, the inflammatory symptoms varying in their intensity, but finally the iris becomes dull and discolored and there is grave danger of sympathetic trouble in the opposite eye. Indeed, sympathetic ophthalmitis in these circumstances has been reported a number of times. *Late cyclitis*—that is, an inflammation occurring after the first week—is characterized by deep-seated circumcorneal injection, thickening and opacity of the capsule, and posterior synechiæ. Under treatment the symptoms may subside or secondary glaucoma may develop.

The *treatment* of these conditions in general terms should include bleeding from the temple by means of leeches, the free use of atropin, dionin, holocain, iced compresses (usually heat is recommended, but iced packs generally are more satisfactory), the administration of *large* doses of salicylate of sodium, and in most circumstances mercury and iodid of potassium. A number of observers have obtained good results from the administration of neosalvarsan. If the process closes the pupil, after the eye becomes quiet iridectomy, iridotomy, or iridocystectomy is indicated.

A remarkable condition to which H. Knapp called special attention is the formation of a *spongy* or *gelatinous exudation* in the anterior chamber, associated at first with considerable pain, congestion of the conjunctiva, and edema of the margins of the lid. The manifestations are those of spongy iritis without inflammation of the iris. The exudation disappears and the results are good, although at first the appearances are most alarming.

Bulging or Cystoid Cicatrix.—Instead of perfectly smooth healing, the cicatrix at the end of a week or two may begin to bulge, sometimes at one or other extremity of the wound, and sometimes through its entire length. The bulging consists in a vesicle-like, semitransparent elevation, and is generally associated with an entanglement of the iris in its margins, together with distortion of the coloboma. Eyes in which such entanglement of the iris has taken place are likely to develop iridokeratitis, and it has been recommended that the cystoid cicatrix should be removed and the opening closed by the application of the electro- or thermocautery. This procedure, however, has proved to be a dangerous one in several instances, and has been followed by severe inflammation and even by sympathetic ophthalmia.

Glaucoma After Extraction.—This complication occurs after a severe iritis, with numerous posterior synechiæ, which has led to the formation of a membrane. It may be the sequel of an iritis, which is characterized by a deep anterior chamber and dotted opacities on the cornea, or also of an iritis which is only slight in character, but where there has been an adherence of the pillars of the coloboma to the cicatrix and also to the lens-capsule, or where tags of capsule have attached themselves to the under surface of the corneal wound (see also page 441). This tends to obliterate the canal of Schlemm. Glaucoma may be

caused by imperfections in technic and by obstruction caused by remnants of capsule and iris, and is prone to follow slow closure of the wound and the formation of an anterior synechia. Sometimes rise of tension develops a few days after operation; sometimes it is a late complication, it may be weeks, months, or even years after the extraction. Elschnig's investigations show that this condition may arise by reason of a proliferation of epithelial cells within the anterior chamber—that is, by an abnormal ingrowth from the anterior corneoscleral surface. Glaucoma of similar origin also occurs after the operation of laceration of the capsule—*i. e.*, after discission. If uncontrolled by miotics, iridectomy, sclerotomy, or cyclodialysis should be performed.

Opacities of the Cornea and Keratitis.—Opacity in the cornea may be due to the introduction of antiseptic fluid, especially solutions of bichlorid of mercury, into the anterior chamber. It has a peculiar, milky-white appearance, and is located chiefly at the posterior surface of the cornea, although the epithelium may also be rough. It does not disappear, and, if sufficiently thick, entirely vitiates the effect of the operation.

This opacity must not be confounded with a very common type of keratitis occurring after cataract extractions, which has received the name *striated keratitis*, consisting of fine stripes of opacity radiating in several directions across the cornea. This disappears in a few days, and need not give rise to apprehension. As Frederick Tooke points out in his admirable studies of the pathology of the corneal section, this condition is more common in elderly subjects than in those of younger years, and may be due to alterations in the endothelium or in Descemet's membrane dependent on age, which permit access of aqueous through the cells, the tension having been released by the corneal section. Occasionally at the end of a week or more *herpes of the cornea*, heralded by sharp pain and lacrimation, may develop, and from the herpetic spots small filaments may arise—*filamentous keratitis*. The lesions will subside under the influence of light bandages and antiseptic lotions; holocain is of much service.

Prolapse of the Iris.—This complication is the chief objection to the operation of simple extraction, and varies in frequency from 3 to 10 per cent., according to different statistics. The prolapse is usually heralded by a sudden sharp pain, which gradually passes away. It generally results from trauma—for example, striking the hand against the eye—or is due to a fit of coughing, violent exertion, straining effort, or similar cause. If the prolapse is discovered soon after its occurrence—that is, at the first dressing—it should be cut off and the edges of the iris reduced, exactly as after the operation of iridectomy. If the prolapse is not noted until the third or fourth day, it is sometimes proper to allow it to remain. The eye should be firmly but gently bandaged and atropin may be instilled, although some surgeons prefer eserin. Small prolapses may disappear, others produce no irritation, while still others become larger, constricted at their bases, or cystoid. H. Knapp allowed these to remain until the irritation had disappeared,

and then amputated them in the same manner as a small staphyloma is abscised, and usually obtained smooth and permanent recovery. Occasionally iridocyclitis occurs, and sympathetic ophthalmitis has been reported. In general terms the safest procedure is to excise the prolapsed iris as soon after its discovery as possible and avoid any permanent entanglement of the iris in the wound which may later be the cause of secondary glaucoma.

Prolapse of the iris after combined extraction—*i. e.*, entanglement of the edge of the cut iris in the angle of the wound—is not uncommon and may also be the determining cause of glaucoma.

Delayed Restoration of the Anterior Chamber and Delayed Healing.—Often the wound after cataract extraction is closed at the end of twenty-four hours, usually not later than the third day. Occasionally, however, there is delayed restoration of the anterior chamber, which in most instances is caused by some foreign substance—for example, a particle of capsule or conjunctiva between the lips of the wound, or to an almost imperceptible incurvation of the flap (Trousseau). Doubtless, in most cases, an error in the technic of making the section is responsible for slow closure of the wound. In a few instances the failure to unite appears to be due to excessive secretion of aqueous humor or to lack of reparative power, depending upon some anomaly in the condition of the patient. Derrick Vail attributes this complication to an involuntary spastic contraction of the orbicularis and has remedied it by severing the muscle, cutting directly upward and downward, from its external attachment. A conservative treatment is generally indicated, and it is usually recommended that bandaging and rest in bed shall be continued until the chamber is restored, but the author agrees with Berry that if any dressing be applied at all, if the wound does not close readily, it should be of the lightest character and should exert no pressure on the lids. Usually removal of the bandage and the adjustment of protecting spectacles (wire-gauze) facilitates the healing (Gifford). If a piece of capsule or other foreign substance can be detected, it should, of course, be removed. A light cauterization of the line of incision with a point of a stick of nitrate of silver or a probe dipped in carbolic acid is often of service. *Delayed union* of the wound, especially after corneal incisions, with separation of its margins, may end in recovery, but has been followed by infection and by glaucoma, especially if the lens capsule becomes adherent to the corneal wound. Associated with tardy or imperfect wound closure there may be a glossy edema of the conjunctiva in its lower part, which H. Knapp called *filtration chemosis*. It will subside when the union of the incision is firm.

Postoperative Delirium and Insanity.—Delirium after operation has been referred to. Sometimes marked dementia follows cataract extraction. The delirium has been ascribed to the use of the bandage, to the effect of atropin, to imperfect mental balance existing prior to the operation, and to auto-intoxication. If possible, the bandage should be removed and the patient given various sedatives, *e. g.*—the bromids

—according to the indications. Paraldehyd is useful; morphin usually does not act well in these circumstances.

Choice of an Operation.—Obviously, the advantages of simple extraction are the absence of mutilation of the iris, and consequently the formation of a round pupil which reacts freely to the changes of light and shade and prevents the dazzling caused by the presence of a coloboma. Its disadvantages are the difficulty of expelling the lens, the increased difficulty of performing perfect toilet of the wound, and the danger of prolapse of the iris. In the judgment of the author certain cases require iridectomy—namely, those in which the ball is hard, the lens is large, the anterior chamber is shallow, the iris is not readily dilatable, or there is ciliary irritation. The combined method is also preferred if the cataract is not ripe or if the patient's mental or physical condition tends to create restlessness. In other circumstances simple extraction may be performed. The author has, however, returned to combined extraction with a small iridectomy, as it is, on the whole, a more satisfactory procedure. For the reasons already given the author is unable to agree with those surgeons who believe extraction of the lens in its capsule should necessarily be the operation of choice, although he fully recognizes the advantages of intracapsular extraction, and has been favorably impressed by the technic developed by A. Knapp (page



Fig. 400.—Knapp's knife-needle.

771). The decision as to which operation shall be chosen must be made according to the character of the cataract and of the eye, and not, as Walter Parker has wisely said, according to any enthusiasm for any one method of procedure. Future studies by means of slit-lamp microscopy may indicate that hernia of the vitreous into the anterior chamber occurs more frequently after extraction in the capsule than after extraction with capsulotomy (see also page 478).

Operations for After-cataract.—After-cataract—or, as it is usually called, *secondary cataract*—has been described. If it is a delicate, web-like membrane which stretches across the pupil, and which is best seen by artificial illumination—*i. e.*, condensing with a large magnifying-glass a beam of light into the pupillary space—the treatment may consist in the introduction of a cataract needle in the manner described under Discission, and making a laceration in the membrane. The operation is readily performed with H. Knapp's knife-needle (Fig. 400) in the manner advised by this surgeon, namely:

The pupil being dilated *ad maximum*, and the area of the operation being perfectly illuminated, the knife-needle is thrust through the cornea 3 mm. from its margin in the horizontal meridian. Next, the knife-needle is advanced to a point close to the opposite margin of the iris, where the membrane is punctured, and a horizontal incision of 4 or 5 mm. is made. This being accomplished, the point of the needle is raised toward the cornea and passed upward in front of the membrane,

which it transfixes at a point 2 mm. above the horizontal incision, and divides it by a cutting movement downward, as far as the horizontal incision. The same procedure is performed on the lower half of the membrane, cutting from below upward. Thus, a crucial incision is formed, and if successful, the retraction of the edges leaves a good central aperture in the membrane. It is essential to cut the after-cataract, and not to tear it nor drag upon the ciliary body. Therefore the instrument should avoid thickened portions of the capsule. It is also desirable that it should not enter deeply into the vitreous.

In place of entering the knife through the cornea in the manner already described, the puncture may be made through the conjunctiva at the corneoscleral border, a method which the author is accustomed to follow. In place of this operation, Ziegler's method (see page 731) may be employed; indeed, in recent years the author has used this admirable operation almost exclusively in all types of after-cataract.

Where the membrane is thick and there has been much proliferation of the epithelium, discission with a knife-needle, owing to the dense and resisting character of the tissues and the danger of dragging upon the

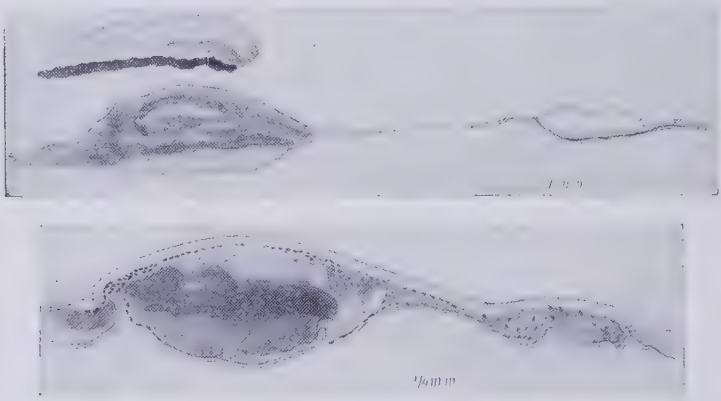


FIG. 401.—Sections of secondary cataract, showing inclusion of cortical remnants between the posterior and anterior capsule and curling of the capsule upon itself. (From a specimen prepared by Dr. C. M. Hosmer in the author's laboratory.)

ciliary body and iris, is a dangerous operation. In these circumstances iridotomy or Ziegler's operation should be employed (see page 731). Extraction of such thickened capsular membranes, as recommended by Col. Smith, is an operation fraught with danger. It has been followed by intra-ocular hemorrhage, by severe iridocyclitis, and also sympathetic ophthalmia.

Other plans are to divide the capsule with delicate cannula scissors, or to cut the desired opening with an instrument which works on the principle of a punch. In place of entering the knife through the cornea or corneoscleral border it may be passed through the sclera 6 mm. behind the corneal margin, and pushed forward so that its point passes through the membrane into the anterior chamber. The membrane is cut by causing the knife to make a sweeping movement from before backward.

Discission is an operation invested with many dangers. In no cir-

cumstances should there be rough handling; the discission instruments must be very sharp, and the operator must avoid dragging upon resisting bands. Preceding the operation and following it there should be the free use of atropin. If signs of reaction occur, the treatment of iritis is indicated.

Glaucoma after discission is an occasional complication, and is characterized by pain, steamy cornea, impaired vision, and increased tension. It should be treated by eserin locally, morphin and chloral internally, and, if these measures fail, by iridectomy or paracentesis and evacuation of the vitreous from the anterior chamber.

In cases of occlusion of the pupil by a drawing up of the iris, or where there are bands of strong inflammatory lymph, to which also the name "secondary cataract" is sometimes applied, discission is not a proper procedure. V-shaped iridotomy with a knife-needle is the best operation (page 731).

OPERATIONS UPON THE EYE MUSCLES

These consist of *complete* and *partial tenotomy* and *advancement* or *readjustment*, and *advancement with muscle resection*. For the operation of tenotomy the following instruments are required: A stop speculum or lid elevator, two strabismus hooks (Figs. 402, 403), fixation forceps, and a pair of probe-pointed scissors, the form devised by Dr. Jackson being particularly suitable. In young children general anesthesia may be necessary; but, if possible, cocain should be used. Usually the internal rectus is divided; quite frequently the external rectus; less commonly the other straight muscles. With rare exceptions the inferior rectus should not be tenotomized.



Figs. 402, 403.—Strabismus hooks.

Complete Tenotomy.—In a tenotomy on the internal rectus, for example, the operator proceeds as follows:

The eyelids being separated with a stop speculum, the surgeon catches with a fine-toothed forceps a fold of conjunctiva and subjacent fascia on a level with the lower border of the tendon, and with the probe-pointed scissors makes an opening just large enough to admit the strabismus hook. He may with one clip divide conjunctiva, subjacent fascia, and the capsule of Tenon; otherwise, after the division of the conjunctiva and subconjunctival tissue, Tenon's capsule must be picked up and incised in a length equal to the cut made in the overlying structures. A strabismus hook is next passed behind the tendon, its point turned upward, and made to appear at the upper border of the tendon beneath the conjunctiva. It is next drawn forward and outward toward the cornea, and scissors, with their blades slightly parted, are introduced between the hook and the eye, and the tendon cut close to its sclerotic attachment. This is the *subconjunctival operation*, and was introduced by Critchett.

Instead, the subconjunctival method, especially in cases where there is a considerable squint, "the open operation" is sometimes per-

formed, but the exposure of the tendon is unnecessarily great and the operation is not as satisfactory as the others recorded.

Snellen's method is as follows:

A small opening, about 4 mm. in width, is made through the conjunctiva over the insertion of the tendon, the center of which is then incised vertically. Through this opening the point of a strabismus hook is inserted and the upper and lower half of the tendon divided. A suture closes the conjunctival wound. Stevens' method, described on page 783, is a modification of this operation and may be used for complete as well as for partial tenotomies.

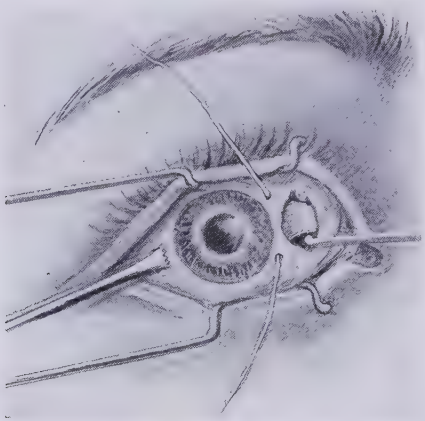


FIG. 404.—Exposure of the internal rectus tendon, which is lifted upon a hook (after Haab). This drawing also illustrates Prince's method of advancement (see page 787). The suture in the sclera, to which the tendon is afterward fastened, is seen lying along the corneal margin.

Tenotomy of the other straight muscles may be performed according to the methods already described, the operator remembering the distance of the insertion of each tendon from the corneal margin (see page 600).

Tenotomy of the Inferior Oblique.—This operation was originally suggested by E. Landolt, but its indications and technic have been especially elaborated by Duane and White.

After injecting the tissues with a 2 per cent. solution of the novocain, a curvilinear incision is made just superior and parallel to the lower and inner bony rim of the orbit, the tissues being divided down to the bone. A strabismus hook is next inserted, and gentle traction is made on the tissues until the muscle is encountered. This is assured by the upward movement imparted to the globe by traction on the hook. The tendon is next drawn forward and divided. The wound having been closed with one or two stitches, a firm dressing is applied and allowed to remain in place for forty-eight hours. Occasionally a subconjunctival ecchymosis appears at the lower and inner part of the globe.

The *indications* for this operation, based upon Duane's studies, are thus summarized by Posey:

(*a*) Complete stationary paralysis of the superior rectus (especially congenital or traumatic) of the opposite eye.

(*b*) Partial paralysis of the superior rectus of the opposite eye if, owing to fixation being performed by the latter, the fellow eye develops a spasm of the inferior oblique, giving rise to diplopia, a disfiguring upshoot of the affected eye, or a lasting torticollis.

(*c*) Spasm of the inferior oblique, either secondary to paralysis of the superior oblique or some other muscle in the same eye, or occurring as a primary condition, provided the symptoms are sufficiently disturbing to warrant operation.

Complications in Tenotomy Operations.—1. The operator may fail to divide the capsule of Tenon. In these circumstances he will also fail to introduce the hook beneath the tendon, and by such failure will recognize that he has not sufficiently incised the tissues.

2. *Hemorrhage.*—Occasionally severe hemorrhage follows a tenotomy, the blood rapidly pouring out beneath the capsule of Tenon and causing alarming proptosis. A firm pressure bandage should be applied, and gradually the proptosis will subside and the blood be absorbed.

3. *Orbital Cellulitis and Tenonitis.*—Cellulitis has occurred from infection of the wound, the inflammation traveling back and causing an inflammation of the tissues of the orbit. The treatment of orbital cellulitis, described in another section, is indicated. Tenonitis, or inflammation of the orbito-ocular fascia, has followed squint operations.

4. *Perforation of the Sclera.*—Although this is a rare accident, it has happened to operators of considerable experience as the result of the use of sharp-pointed scissors, and for this reason the probe-pointed instrument is always to be preferred. In such circumstances the eye should be treated in the manner described on page 328.

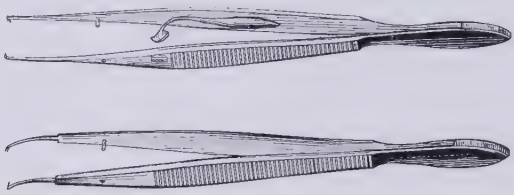
5. *Retraction of the caruncle,* so that it sinks away from its normal position and gives a most disagreeable and peculiar stare to the eye, is a very unfortunate occurrence after a squint operation. A very slight degree of this is liable to occur even after the most careful tenotomy of the internus. Where it exists in great degree, it is due in part to excessive dissection of the tissues, and in part to retraction of the muscle. There are several methods of overcoming this defect, the essential character of which is the loosening up of the contracted tissues and stitching the caruncle into place.

Partial or Graduated Tenotomy.—Graduated tenotomies are performed for the purpose of correcting those conditions which are described under Heterophoria. The operation was especially elaborated by Dr. Stevens, of New York, and is performed as follows:

With a pair of small, narrow-bladed scissors a transverse incision is made through the conjunctiva exactly corresponding to the line of insertion of the tendon. This is seized behind, but near its insertion, and a small opening is made dividing the center of the tendinous expansion exactly on the sclera. This opening is then enlarged by careful cuts with the scissors toward each edge, keeping carefully on the sclera as the border of the tendon is approached; the amount to be cut depends upon the judgment of the operator and the need of the case, and is further regulated

by placing the patient before a lighted candle and testing the sufficiency of the muscle upon which the operation is made, in the manner already described in connection with the investigation of heterophoria. In dealing with strabismus, the surgeon may determine to continue his section through the border, leaving uninjured, as far as possible, both the anterior and posterior lamellæ of the capsule, as well as the expansion at each border, to hold the muscle in relation to the eye. Turning the scissors then in the direction of the other border, this portion is dissected with equal care.

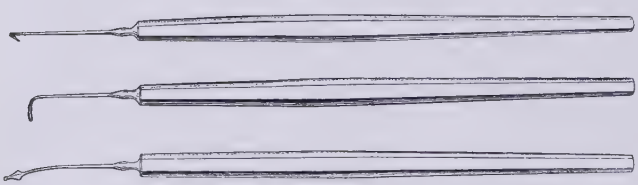
Figures 405 to 410 illustrate the delicate instruments which are used in this operation. They may with equal propriety be employed in ordinary tenotomies, and are satisfactory for this purpose, inas-



FIGS. 405, 406.



FIG. 407.



FIGS. 408–410.

FIGS. 405–410.—Stevens' instruments for tenotomy.

much as the laceration of the tissues is less marked, while the effect is equally great if the incisions are carried sufficiently far according to the directions already given.

Several other operative procedures for the purpose of elongating the tendon by partial tenotomies have been designed, especially by Ziegler, Verhoeff, and Todd. The first-named surgeon describes the characteristics of his operation as "complete division of each lateral third of the tendon and careful snipping of the superficial fibers in the central third, until sufficient elongation is obtained to yield a measurement of orthophoria."

Advancement or **readjustment** is an operation in which the tendon of a rectus muscle is brought forward to a new attachment.

The operation is applicable to cases in which the tendon has become weakened, as, for instance, in myopia, together with the production of divergent squint; to those cases of convergent strabismus in which it is desirable to combine advancement of the external rectus with tenotomy of the internus; to free bilateral advancement to the exclusion of tenotomy; to certain cases of heterophoria (see page 645); and to cases in which an injudicious division of the internal rectus, for instance, has converted a convergent into a divergent squint. For other indications, see pages 635 and 636. General anesthesia may be necessary in young subjects and nervous patients.

The same instruments which are used in tenotomy are required, in addition to which suitable curved needles, a needle-holder, silk thread, fine catgut, and advancement forceps should be provided. Numerous methods of advancement have been designed. It is possible only to record in detail a few standard operations and to make reference to others that have proved their value. With the operation, a description of which follows, somewhat modified from the one which was recommended by Swanzy, the author has achieved satisfactory results:

An opening is made in the conjunctiva and capsule immediately over the insertion of the tendon which is to be advanced, twice the breadth of the tendon. A band of conjunctiva between the opening and the cornea is next separated with the scissors from the sclera. A strabismus hook is now passed under the tendon, which is freely separated from the sclera; the hook is brought well up to the insertion of the tendon, care being taken that the whole width of the tendon is held on the hook, and that by lifting it the under surface of the muscle is exposed. A curved needle carrying a strong black silk suture is introduced from its upper margin between the muscle and sclera, and passed through the muscle at its outer third. In the same way another suture is passed behind the muscle from its lower margin. Each of these sutures is fastened on the muscle with one hitch of a surgeon's knot, a long end being left to each. For the strabismus hook Prince's advancement forceps is now substituted, which firmly grasps the tendon, which is next separated with scissors from the sclera close to its insertion. The needle on the end of each suture is next passed through the superficial layers of the sclera and beneath the conjunctival flap to the margin of the cornea in the manner illustrated in Fig. 412, and while an assistant rotates the eyeball toward the muscle which is to be advanced, each suture is tied with its own end. The redundant tissue is trimmed away and the conjunctiva sewed with three interrupted sutures over the advanced muscle, the central suture being passed through the conjunctiva, capsule and the advanced muscle to the margin of the cornea. Naturally, a greater or less effect is produced according as the sutures are placed farther from or nearer to the tendon, and according to the extent to which the loosened muscle is drawn toward the corneal margin. Both eyes are bandaged and should remain covered for at least four days, when the superficial sutures are removed. The deep sutures are allowed to remain, if they produce no irritation, from eight to ten days. An objection to this operation is the knot which must be removed through a small opening in the conjunctival surface, but if black silk is used the author has not experienced any difficulty; and it has seemed to him that the results were better than if the suture was not fastened in the manner described.

Worth's Method of Advancement.—A curved vertical incision, 12 to 14 mm. in length, is made through the conjunctiva, with its convexity close to the cornea, followed by a similar incision through the capsule of Tenon. If the capsule and conjunctiva do not retract, they are pushed back so as to expose the insertion of the tendon. Next, one blade of an advancement forceps is accurately passed beneath the tendon, and the other blade, which is superficial to all the structures, is

attached so that tendon, capsule, and conjunctiva are all firmly held together. The tendon is now divided by means of scissors at its insertion into the sclera, and the area of the sclera near the cornea destined for the new insertion of the advanced tendon is carefully cleaned of all loose tissue. By raising the advancement forceps, holding the conjunctiva, capsule, and tendon, the underside of the muscle can be examined. A suture is now passed from the conjunctival surface exactly through the center of the muscle to its under surface and carried under the advancement forceps to the corneal margin, where it is inserted into the sclera in exact alignment with the old insertion of the tendon; this is a "marking suture."

Next, a needle carrying a suture, previously greased with sterile vaselin, is passed from without inward through the conjunctiva, capsule, and muscle, and again through the muscle, capsule, and conjunctiva, so that the bight of the suture shall include a width of 2 mm. near the edge of the muscle, together with the capsule and conjunctiva. The ends of the threads are now crossed over, making a half-hitch, and the

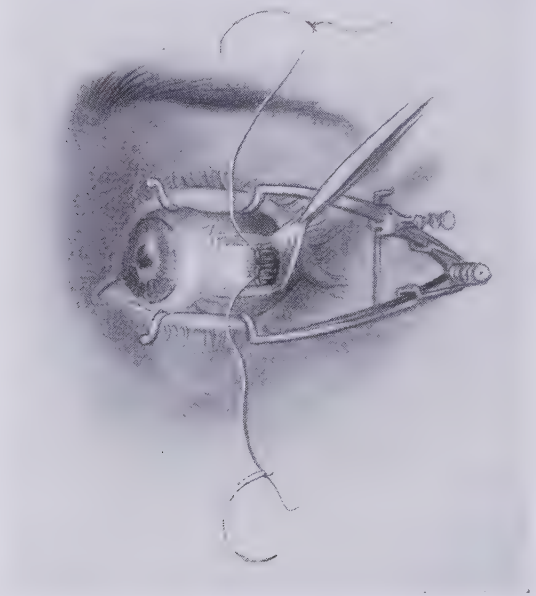


FIG. 411.—Advancement of the external rectus: The muscle has been exposed and the sutures tied upon it (page 785).

needle carrying the suture is passed through the conjunctiva, capsule, and muscle, and carried beneath the lower blade of the advancement forceps, out of the wound, and stuck in a small piece of gauze laid on the patient's forehead. Precisely the same procedure is repeated at the lower margin of the muscle at a point exactly opposite to that of the first suture.

The anterior parts of the muscle and capsule and conjunctiva are next removed by cutting them with scissors just behind where they are grasped by the advancement forceps. The sutures on the muscle should be at least 3 mm. behind the cut end. The next stage includes the insertion of the two main sutures into the sclera at the corneal margin. A firm hold of the globe is taken at the site of the old insertion of the tendon. The needle carrying the upper suture is laid on the sclera exactly in line with the "marking suture," that is, parallel to it, about 4 mm. from the corneal margin, pressed backward in order slightly to dimple the sclera, and then pressed onward so that it shall traverse about half the sclera, but must not penetrate

its whole thickness, the superficial extent of the suture being about 3 mm. It is pushed onward until it can be grasped with the needle-holder just below its point and pulled through. The other (lower) main suture is manipulated in precisely the same manner. The eyeball is next rotated into the primary position, and each suture tied with the first hitch of a surgeon's knot, and gradually tightened, while the anterior end of the muscle is brought into the desired position and the knot completed by a second hitch. The marking suture is next tied, and, if any gaps remain, fine sutures are used to close them.

In A. E. Prince's method of advancement an unyielding fixation point is obtained by utilizing the dense episcleral tissue, severing the muscle, and regulating the effect by a "pulley suture" (Fig. 404).

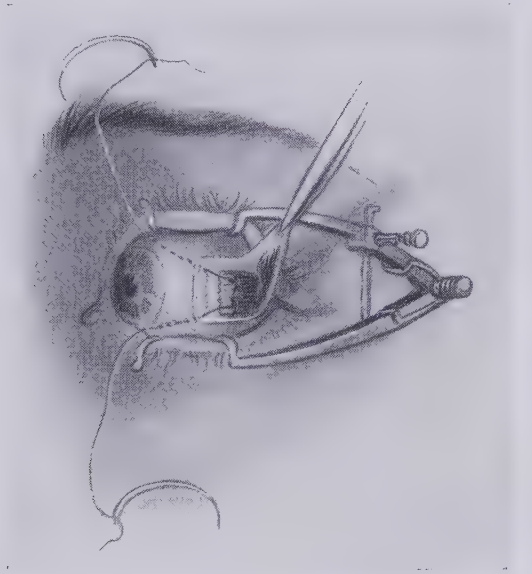


FIG. 412.—Advancement of the external rectus: The tendon has been separated from its scleral attachment, and the sutures will be passed through the scleral tissue beneath the conjunctiva in the direction of the broken line and the muscle advanced to the corneal margin (page 785).

H. D. Bruns, of New Orleans, has described an ingenious operation for advancement of the recti tendons, performed with the aid of a Clark hook and the formation of a tuck in the tendon, which is firmly flattened down and drawn strongly forward, and held in place by a combination of pulley and guy suture. In Landolt's advancement operation after exposure of the tendon of the muscle to be advanced, the operator passes the two sutures, from without inward, about one-third of the width of the muscle from each edge; they include the surrounding tissue and are made to traverse the episcleral tissue so as to advance the tendon to the corneal margin.

Greenwood performs a *combined tucking* and *advancement* in that after exposure of the muscle an ordinary tuck is made and a suture passed through each corner of the top of the tuck and next through the

episcleral tissue in such a manner that the tuck is fastened down to the sclera well forward. Finally, the conjunctiva is sutured over the muscle thus tucked and advanced.

Meyer Wiener in his advancement operation ties the sutures over flat metal (gold) plates on the same principle as the tension sutures used in harelip operations. Lancaster exposes the muscle to be advanced by a straight longitudinal incision from near the cornea to near the canthus; a longitudinal incision is also made in the capsule. The muscle sutures are in the form of a whip-stitch enforced by a security stitch.[1]

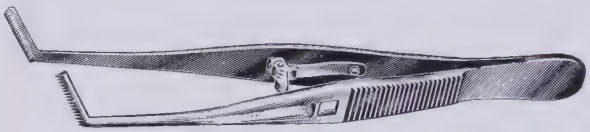


FIG. 413.—Reese's forceps for operation in squint.

Of the methods of advancement which have been recorded, the author is in the habit of employing the one which is described first; Landolt's and Worth's operation have also given him satisfaction.

Reese's Muscle-resection Operation.—Dr. Robert G. Reese operates as follows, the description of the operation being in his own words. The method is much employed and by many surgeons almost to the exclusion of any other procedure.

For the external and internal rectus make a vertical incision in the *conjunctiva* 6 mm. from the corneoscleral margin, commencing at the level of the upper corneal border, and extending to the horizontal plane of the lower border.

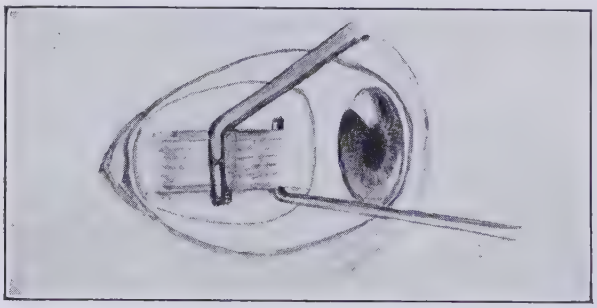


FIG. 414.—Passage of the strabismus hook under the entire muscle (Reese).

At the upper and lower limits of the incision just made grasp the tissue anterior to the sclera with forceps and open with scissors, directing their point away from the muscle. This procedure allows the passage of the strabismus hook under the entire muscle.

When the muscle is held on the hook, dissect all the conjunctival and subconjunctival tissue back to the canthus, exposing the bare muscle completely.

The lateral invaginations of Tenon's capsule, which are attached to the tendons of the ocular muscles, must be dissected free and clear.

[1] American Journal of Ophthalmology, March, 1918. Well illustrated.

One blade of the resection forceps is then inserted beneath the muscle at a right angle to its course, so that the groove on the blade lies directly over the middle fibers of the muscle. Clamp the forceps to the last notch, and do not let its grasp include anything but muscle.

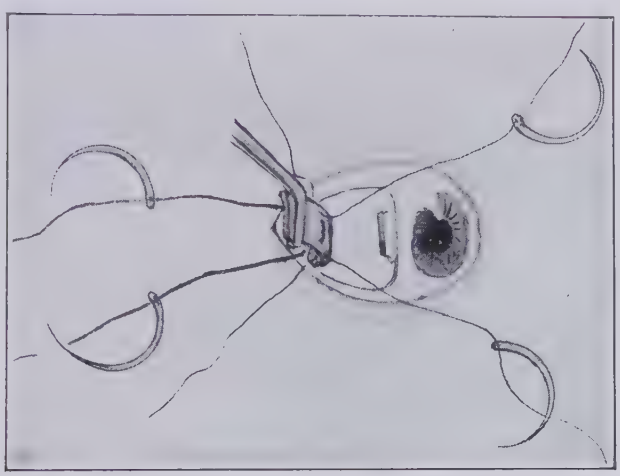


FIG. 415.—Showing the three necessary sutures (Reese).

Sever the muscle 2 mm. from its scleral attachment, leaving a stump, so that the resected end can be sewed to its original insertion. Free the belly of the muscle from any scleral adhesions. Three sutures are necessary.

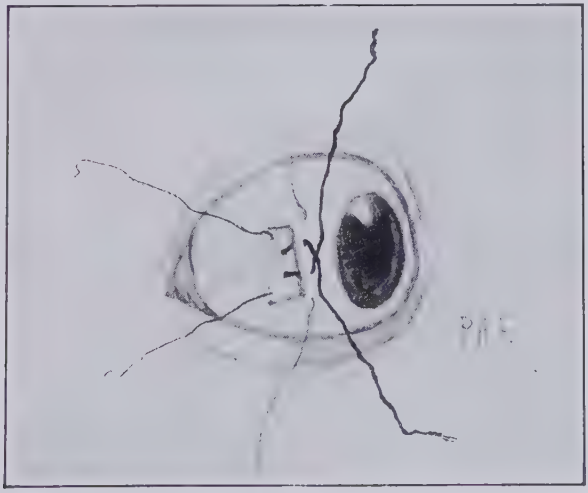


FIG. 416.—Showing the muscle resected and sutures in scleral stump (Reese).

Put the sutures in, commencing with the middle, which is a No. 3 braided silk, with a needle on each end. Pass one needle through the scleral surface of the muscle posterior to the blade of the forceps and 4 mm. back of the point of resection, and 1 mm. to the side of the groove on the forceps; then pass the other needle the same

way, but to the other side of the groove, making a loop with the suture on the scleral surface of the muscle. As the needles pierce the muscle, let them include the dissected edge of subconjunctival and conjunctival tissues.

The two wing sutures are No. 5 silk with a single needle passed *first* through the upper and lower part of the dissected conjunctiva and episcleral tissues, including the superior and inferior border of the muscle, and slightly posterior to the loop made by the middle suture.

Cut the muscle anterior to the sutures, leaving at least 2 mm. in front of the loop.

Insert the two needles attached to the middle suture 2 mm. apart, through the center, and the other two needles through the upper and lower edges of the scleral stump. These needles should include the conjunctiva as they pass from behind forward.

Tie the middle suture first in a loop, and do not use a surgeon's knot, as it will not pull up well. The lateral sutures are next tied. No supplementary conjunctival sutures are necessary.

The middle suture is removed in ten days, and the others can be taken out any time after forty-eight hours, or if left in they soon fall out.

The eye operated upon only is bandaged and is dressed daily for five days, when boric acid bathing, three times a day, is ordered; and if the eye is not overcorrected, the correcting lenses are ordered for constant use. On the other hand, if there is an undercorrection, a mydriatic is used and the proper glass is worn constantly.

The operation consists in resecting the muscle only, and not cutting out any of the other tissues of the eye. It is a myectomy, because in no degree of squint, however slight, will removing the tendinous portion of the muscle be sufficient.

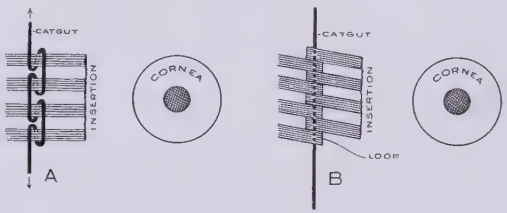


FIG. 417.—"Cinch shortening" operation by O'Connor.

O'Connor's "Cinch Shortening" Operation.—The condensed description has been kindly furnished by Dr. O'Connor. The operation is one in which no sutures are used, the method of shortening a saddle girth (cinch), employed in our cavalry service, being adapted to the ocular tendon. The cut shows, in a diagrammatic manner, the technic.

"After exposure of the tendon, including stripping back of its capsule, it is split into a number of bands, by blunt separation, each about 4.5 mm. in width. The shortener, which is carried on a heavy curved needle, is passed over each tendon band as shown at A. A tenotomy hook is then placed under the 'shortener' at one tendon margin, and the corresponding end pulled against the hook. This causes the tendon to loop itself about the 'shortener.' This maneuver may have to be repeated at the other tendon margin. When the bands are all looped the appearance is as at B. The material now used as the shortener is known as 'dermal suture and tension ligature.' One end is left long enough to protrude through the conjunctival wound. In this way it acts as a drain to the seat of shortening and facilitates removal, which is done in from ten to fourteen days. The following advantages are claimed: (1) No possible loss from slipping, (2) tenotomy of opponent not required unless it is contracted, (3) early use of both eyes prevents the site of

operation from forming adhesions to the globe, thus preserving the normal insertion, (4) permits operations on small children (three to four) without fear of later overeffect, (5) permits a very accurate correction of vertical deviations."

Advancement of the capsule of Tenon is recommended by some surgeons. S. Lewis Ziegler has designed a *capsulomuscular advancement with partial resection.* His method is as follows:

A vertical incision is made in the conjunctiva near the limbus and the tendon and muscle raised on two tenotomy hooks introduced from below upward and put slightly on a stretch. One arm of a double-armed suture is then inserted through the lower edge of the muscle from before backward and the same suture repeated behind the first, thus making a marginal whip-stitch. The thread is then passed partly across the top of the muscle and a similar double stitch is duplicated on the upper margin of the muscle.

Each needle is now carried backward in a parallel line beneath Tenon's capsule and made to emerge on the conjunctival surface. A small wedge or V-shaped piece is then cut away from each peripheral third of the muscle, above and below, with punch or scissors, leaving a central strand of fibers intact.

The needles are now entered through the conjunctiva at the upper and lower extremities of the wound, passed firmly into the sclera for solid anchorage, and brought out near the limbus, where the suture is tied after the toilet of the wound has been performed. This leaves a knot and two parallel lines of thread exposed on the conjunctival surface, thus holding all the tissue flat against the sclera. The good judgment of the operator must decide how much capsule shall be engaged and just how tight the graduated suture shall be drawn. As a rule, the squint should be slightly overcorrected. If indicated, certain measures should also be taken to weaken the pull of the antagonist.

Tendon Transplantation.—As Jackson points out, "operative transfer of the function of one muscle to another offers a remedy for defects of movement which cannot be overcome by nerve impulses acting through the usual channels." Such operative procedures are constantly employed in the correction of ptosis (page 69) and also have been used in cases of paralysis of the superior oblique, of the oculomotor, and especially of the external rectus. To obviate the diplopia caused by paralysis of the superior oblique the insertion of the superior rectus may be placed more posteriorly and also moved to the temporal side of the sclera (Jackson's method). In general terms, in paralysis of the external rectus the outer halves of the superior and inferior recti are separated from their own insertion, brought down, and sewed into the external rectus at its insertion; in paralysis of the internal rectus the inner halves of the vertical recti are used and attached in like manner to the internal rectus at its insertion; in paralysis of the superior rectus the upper halves of the horizontal recti may be transplanted (O'Connor), and in paralysis or loss of action of the inferior rectus transplantation of fibers from the internal and external recti may be done (Posey).[1]

Muscle Recession with Scleral Suturing.—To this operation which in general terms is the reverse of an advancement, renewed at-

[1] For the details of operative technic original reports should be consulted. See Jackson, E., Transfer of Function of Ocular Muscles, American Journal of Ophthalmology, 1923, 6, No. 2, p. 117. A full bibliography of important papers on this subject is appended.

tention has been recently called by P. Chalmers Jameson, who has elaborated[1] a technic which has yielded him satisfactory results in the correction of concomitant strabismus. The operator freely exposes the muscle and, after detaching it, separates it from the globe, as well as its capsular continuity above and below, but leaves its sheath intact. The detached muscle is transplanted to a position, carefully determined by measurement, further back on the sclera, where it is secured by sutures.

OPERATIONS UPON THE LACRIMAL APPARATUS

Slitting the Canaliculus.—This is performed as follows:

The lid being drawn down and out with the thumb, and the canaliculus knife held vertically, the probe point is introduced into the punctum. The handle is now depressed into the horizontal position, and the instrument pushed along the canal



Fig. 418.—Weber's canaliculus knife.

until the probe point touches the inner wall of the lacrimal sac. It is then raised to the vertical line with the cutting blade turned slightly inward, and the roof of the canaliculus divided. Either the upper or the lower canaliculus may be slit.

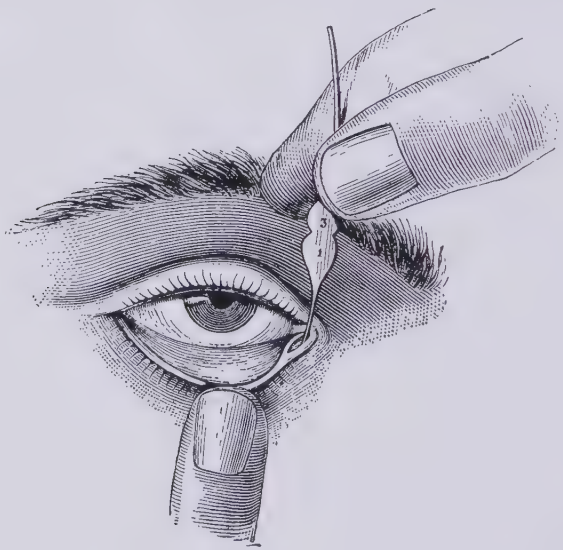


Fig. 419.—Introduction of a lacrimal probe (Meyer).

Introduction of the Lacrimal Probe.—The probe (Bowman's or Williams' probes are commonly employed; modifications have been devised by Theobald and Tansley) is introduced by passing it hori-

[1] For the method of operating as advocated by Jameson, see Archives of Ophthalmology, 1922, vol. li, p. 421.

zontally along the canaliculus until its point touches the lacrimal bone. It is raised to the vertical position and pushed into the duct, remembering that the direction should be downward, slightly backward, and usually outward. Ziegler performs *rapid dilatation* of the lacrimonasal duct with a specially devised *dilator*.

Incision of a Stricture.—If the stricture resists, it may be divided with a knife, either the one which has been employed in slitting the canaliculus or, still better, with the instrument of Stilling. The knife is introduced in the same way as the probe, pushed down into the duct, and the stricture incised. The knife is next partially withdrawn,

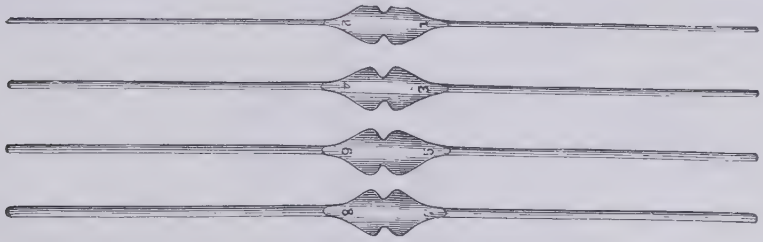


FIG. 420.—Lacrimal probes.

turned slightly, and the maneuver repeated. Dr. Charles Hermon Thomas devised a special knife, or *stricturotome*, which may be utilized for this purpose.

Introduction of the Lacrimal Syringe.—The nozzle of an Anel syringe can be introduced along the canaliculus without slitting it. The lid is drawn down and outward in the same manner as if the operation of slitting the canaliculus were to be performed, and the point of the syringe introduced. Sometimes the punctum is swollen shut and the nozzle cannot be inserted. In these circumstances the punctum may be dilated with a silver pin. Ordinarily a lacrimal syringe is



FIG. 421.—Thomas' stricturotome.

furnished with a cannula probe. This is introduced into the duct in precisely the same manner as the solid probe; the syringe is filled with an antiseptic fluid, inserted into the mouth of the cannula, and the liquid injected into the duct.

Curetage in Dacryocystitis.—W. R. Thompson in the treatment of *dacryocystitis* slits the canaliculus, introduces a small sharp curet with which the sac or the duct, or both, according to the condition, are carefully but thoroughly cureted. Following this a 25 per cent. solution of iodin is applied to the inner walls of the sac and duct. John Green, Jr., after freely slitting the canaliculus, performs rapid dilatation

of the lacrimonasal duct with a Theobald probe (No. 5 or 6) and follows this by curetage according to the method of Thompson. These surgeons report satisfactory results. With their technic the author has had no practical experience.

Excision of the Lacrimal Sac.—In order to meet the indications described on page 657, excision of the lacrimal sac may be performed as follows:

After thorough cleansing of the sac through the canaliculus with a 1 : 10,000 bichlorid of mercury solution, general anesthesia may be induced, although in most

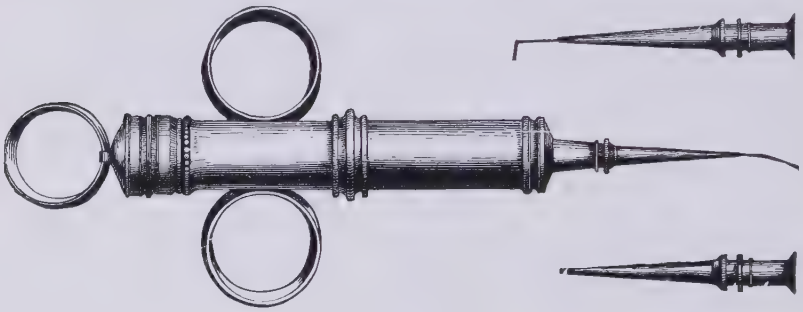


Fig. 422.—Anel syringe.

nstances careful local anesthesia will be sufficient (see page 689). With the skin drawn toward the bridge of the nose, the surgeon makes a slightly curved incision down to the periosteum, which extends from 4 mm. above the internal palpebral

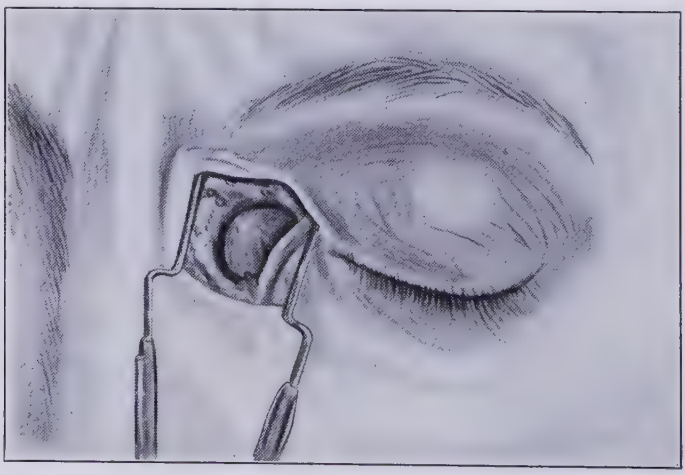


Fig. 423.—Extirpation of the lacrimal sac (Haab).

ligament to 5 mm. below it, its length being 2½ cm. The canthal ligament may or may not be divided with scissors, and while the lips of the wound are separated, the temporal lip being especially drawn outward, the fibrous expansion from the tendo

oculi is divided through its whole length, exposing the sac, which usually can be recognized by its bluish color. The sac is next gradually separated from the periosteum, being dissected out very much in the manner of removing a cyst, care being taken not to rupture its walls. The internal surface, the upper end and the posterior surface of the sac having been freed, is cut through at the commencement of the nasal duct. Sometimes the field of observation is obscured by a smart hemorrhage, which usually can be controlled by pressure or by specially devised specula; for example, those introduced by Axenfeld, or even more satisfactorily with the retractor designed by Bishop Harman. Should the operator experience any difficulty in outlining the sac, its position may be localized by inserting a strabismus hook through the canaliculus into the sac and keeping it there during the operation. This method was introduced by E. A. Shumway, and is most satisfactory in practice. Some surgeons advise that the sac shall be filled with melted paraffin prior to the operation, a procedure which the author has never found to be necessary.

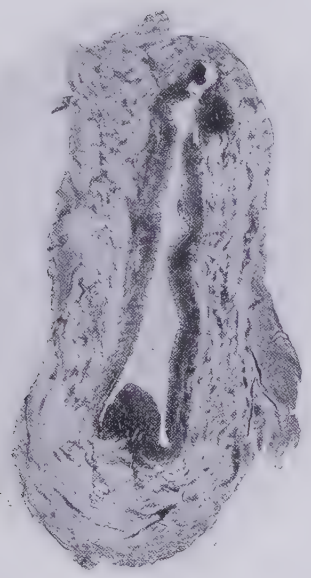


FIG. 424.—Excised lacrimal sac. Dense infiltration of mucosa with round-cells; erosion and degeneration of the epithelium; sac wall densely fibrous and vessels engorged. Insane patient (Philadelphia General Hospital).

C. R. Holmes did not believe that division of the tendo oculi is required in order to expose the sac, but dissected out the sac from underneath the tendon. If the tendo oculi has been severed, it may be replaced or repaired by a strong suture. Great care must be taken that every portion of the sac is removed, and the operation may be terminated by thoroughly cureting the region (which usually is unnecessary if the technic has been correct) of the ductus ad nasum, removing all traces of mucous membrane. Two sutures close the wound, which usually heals promptly. Holmes advises that the canaliculi should also be destroyed. Otherwise a blind pocket forms at the inner canthus. In order to accomplish this he split the canaliculi through their entire length and destroyed their lining membrane with the actual cautery. The dressing should consist of a pressure bandage placed over a light compress. There are many modifications of the operation of excision of the lacrimal sac. The most elaborate technic is the one devised by Meller.

Extirpation of the Lacrimal Gland.—Following the direction of C. R. Holmes, this may be performed as follows:

An incision beginning near the center of the upper orbital arch and following the bony margin is carried to a point 3 mm. below the outer canthus. Next the fascia or septum orbitale is cut through along its attachment to the orbital margin. Should fatty tissue be present in the wound, it must be held to one side with retractors and all bleeding from the edge of the wound must be controlled before the gland is separated from its surroundings, inasmuch as it is sometimes very difficult to distinguish the gland from the surrounding fatty tissue. By means of blunt-pointed scissors, fixation forceps, a small knife, and tenotomy hooks the dissection of the gland can be accomplished, and it may be removed without leaving any portion of it behind. Before the wound is closed all bleeding must be stopped. The lips of the wound are united with interrupted silk sutures, and the usual antiseptic dressing applied. As complications, hemorrhage into the orbit and atrophy of the optic nerve have been reported, and on a number of occasions a persisting conjunctivitis, and also, as, for example, in Veasey's case, a form of keratitis.

Extirpation of the Palpebral Portion of the Lacrimal Gland.—Instead of the removal of the orbital lacrimal gland, extirpation of the palpebral gland is often practised. It is a much simpler operation and may be performed as follows:

Thorough local anesthesia having been secured, the upper lid is everted and drawn upward from the eyeball while the patient looks strongly downward. This exposes the palpebral gland, which may be seized with toothed forceps and drawn outward. Its conjunctival covering is next incised, and the gland dissected from its surroundings. Hemorrhage having been controlled, the wound may be closed with one or two interrupted silk or catgut sutures, the upper lid replaced, and a light pressure bandage applied. The stitches are removed on the third day.

Toti's Operation (*Dacryocystorhinostomia*).—In this operation a passageway for the tears is made through the bony wall of the nose, and its main points are briefly summarized by Török, who recommends it as a valuable procedure, as follows:

After chiseling through the bony lacrimal fossa, the internal wall of the lacrimal sac is resected, and at the same time a part of the mucous membrane of the nose is removed, which is of approximately the same size as the remaining external wall of the sac. The edges of the two mucous membranes are now brought into close connection, so as to permit their growing together. The mucous membrane of the lacrimal sac takes the place of the removed nasal mucous membrane. Together they now form one continuous mucous membrane, and with that the closing of the defect is prevented. The author has had no experience with this operation.

West's Operation (*Window Resection of the Nasal Duct in Stenosis*).—This consists in resecting, under local anesthesia, a window from the nasal duct in the upper part of the nose above the inferior turbinate, and involves the removal of part of the lacrimal bone and also a piece from the superior maxilla. This removes a stricture in the upper part of the duct, but leaves a stenosis in the lower part untouched. It is, according to J. M. West, who has designed this operation and whose description is quoted, immaterial whether the lower part of the duct is stenosed, so long as the tears can drain through the artificial window. Previous to operation a probe is passed into the duct to act as a guide.

Mosher's Operation.—This is an operation devised for the purpose of draining the lacrimal sac and the nasal duct into the unciform fossa. Mosher summarizes the technic as follows:

"The essential steps of the operation are the uncovering of the unciform fossa by the removal of the anterior end of the middle turbinate, and the dissection of a mucous membrane and periosteal flap from the fossa: the breaking down of the inner wall of the unciform cell and the slitting of the inner wall of the nasal duct and lacrimal sac, and the widening of the nasal duct by removing the lip of the ascending process of the superior maxilla. The thing to avoid is opening the unciform groove and establishing an accessory ostium of the antrum which, from its position, would carry infection from the lacrimal sac into the cavity of the sinus. Like all other operations on the lacrimal canal, it faces the danger of cicatricial closure of the canal and the necessity of reslitting the duct and the sac; but unlike the other measures it gives a much larger opening of the lacrimal sac and the nasal duct. Should reslitting of the canal become necessary, it is easily executed under direct vision. The operation has the advantage that the manipulations are carried out in thin bone and toward an absolute anatomic landmark."[1]

Recently Mosher has advocated and successfully practised a combined intranasal and external operation on the lacrimal sac (Mosher-Toti operation). The technic of this operative procedure is fully described and illustrated by Mosher (see footnote reference).

With endonasal operations for the relief of chronic dacryocystitis the author has no practical experience. His results from excision of the sac have usually been satisfactory. The resulting epiphora often subsides materially because of the elimination of the irritating influence of purulent secretion; sometimes it practically disappears. Many surgeons, however, are dissatisfied with extirpation of the sac, and advocate the endonasal operation. It would seem that the combined method is far superior to the endonasal route.

[1] The treatment of chronic dacryocystitis from the standpoint of intranasal drainage has been discussed by a number of other surgeons and various additional operations have been designed. Consult: J. V. Patterson and J. S. Frazer, British Journal Ophthalmology, iii, 1919. Yaukauer, The Laryngoscope, xxii, 1912. W. L. Benedict and R. A. Barlow, American Journal Ophthalmology, vol. ii, 1919. H. P. Mosher, The Combined Intranasal and External Operation on the Lacrimal Sac (Mosher-Toti), Ann. of Otol., Rhinol., and Laryngol., March, 1923.

APPENDIX

The Use of the Ophthalmometer.—Ophthalmometry or, more properly, keratometry has been briefly referred to on page 120. A number of new models of the Javal-Schiötz ophthalmometer are now obtainable, with variations in the disk, form of arm, and method of illumination, but they do not introduce radical changes. The following rules, prepared by the late Dr. E. W. Stevens, formerly associated with the author in the Philadelphia Polyclinic (now the Polyclinic Section of the Graduate School of Medicine of the University of Pennsylvania), will enable the student to understand the proper

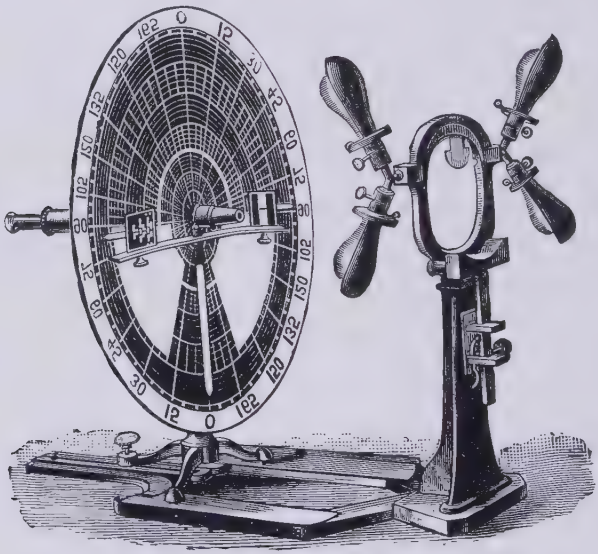


FIG. 425.—Javal-Schiötz ophthalmometer (old model).

method of using this instrument, as it is illustrated in Fig. 425, as well as, in general terms, of the modern models.

The examiner, after satisfying himself that the illumination from the electric bulb is accurate, should carefully adjust the telescope by looking through it and turning the eye-piece either to the right or the left until the cross-hairs are brought clearly into view. The telescope is then turned so that the long pointer is below and at zero. The stationary mire on the parallelogram (Fig. 426, *A*) should be examined to see that it is in proper position, which is at 20° on the graduated arc.

The patient is now seated before the instrument in an easy position, with his chin resting on the chin-rest and his forehead pressed against the forehead-rest. His eyes should be widely opened and exactly horizontal—points to be determined by sighting through the transverse slit above the telescope. One eye is now covered with a small shade, and the observer sights along the telescope, through the notch above it, at the patient's eyebrow; then, sighting through the tube, he moves the

instrument forward or backward and raises or lowers it by the thumb-screw until the eye is brought into the field of the telescope, and a distinct image of the disk and mires is seen on the cornea.

The images of the disk are doubled, and, overlapping each other, form an oval space in which are seen the two mires or targets, to which the beginner should confine his attention. The observer now slides the mire at his right along the arc until its reflection touches the reflection of the stationary mire, and notes whether the two lines bisecting the two mires are continuous. If these two lines are not continuous the telescope is turned so that the long pointer will move from 0° toward 135°. If the transverse lines do not become continuous when 135° is reached, the rotation proceeds no farther in this direction, but the long pointer is turned back to 0° and then toward 45°, but never beyond 45°. With regular astigmatism the lines always become continuous within 45° of 0°. When the lines are continuous the mires must be brought into perfect approximation (Fig. 426, 1). This is the *primary position*, which should be carefully recorded according to the position of the long pointer.

The telescope is next turned so that the long pointer moves 90° to the left of the primary position—*i. e.*, to a point which is known as the *second position.*

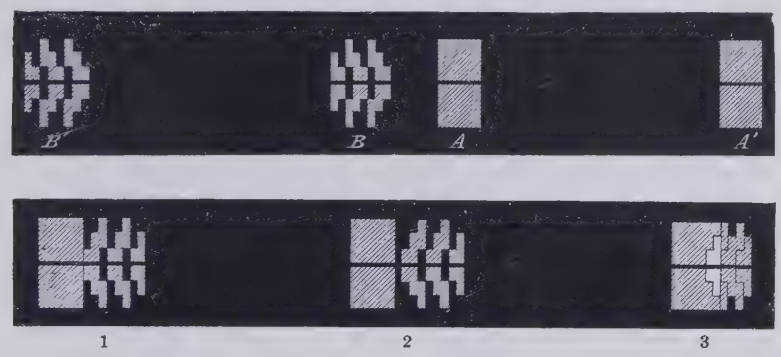


FIG. 426.—The mires.

If the mires overlap (Fig. 426, 3)—for example, two steps in the second position with the long pointer at 90°—there is astigmatism of 2.00 D with the rule, because each step is equivalent to 1 diopter of corneal refraction, and this is recorded + 2.00 D cyl., axis 90°, or —2.00 D cyl., axis 180°.

If, on the other hand, the mires separate (Fig. 426, 2) in the second position, there is astigmatism against the rule. For example, if the primary position is found at 30°, and when the tube is turned to the left until the long pointer reaches 120° a separation of one step has occurred, there is astigmatism of one diopter against the rule, which is recorded + 1.00 D cyl., axis 30°, or —1.00 D cyl., axis 120°.

In order to ascertain the exact number of steps to which the separation of the mires in the second position is equivalent, they are approximated by moving the sliding mire until the reflections touch, and the telescope is then rotated back to the primary position. The mires will now overlap, and the amount of astigmatism can be read off just as in astigmatism with the rule. The observer should remember, in finding the primary position, not to turn the long pointer farther than 45° on each side of 0° at the lower margin of the disk, lest he record astigmatism against the rule when it is with the rule, and vice versa.

The upper surface of the arc carrying the mires is graduated on its outer circle to show diopters of refraction. It does not give the hyperopia or myopia of the eye, but indicates the corneal curvature. On the clamp of each mire there is a mark which enables one to read at a glance from this graduated arc the total refraction of each meridian of the cornea. The total refraction of at least one corneal meridian

should be recorded, and preferably the one of least refraction. For example, if the examiner finds in the right eye 1 diopter of astigmatism with the rule, the long pointer being at 75° in the second position, and the right-hand mire at 23° on the graduated arc, the refraction may be recorded O. D. 43.00 D = 1.00 D cyl., axis 75° with the rule.

If so desired, the astigmatism can be read from the graduated arc by measuring alternately the meridians of greatest and least refraction of the cornea.

On the right of the inner circle of the arc there is a scale graduated from 6 to 10, each space being divided into ten equal parts. These spaces record the radius of curvature of the cornea in millimeters, and the amount is indicated by a mark on the clamp of the traveling mire.

In some eyes it is impossible to bring into a continuous line the two lines bisecting the mires of the ophthalmometer, owing to irregular astigmatism or conical cornea. In these cases, however, the instrument is perhaps superior to all other methods of corneal measurement, as the overlapping or separation of the mires

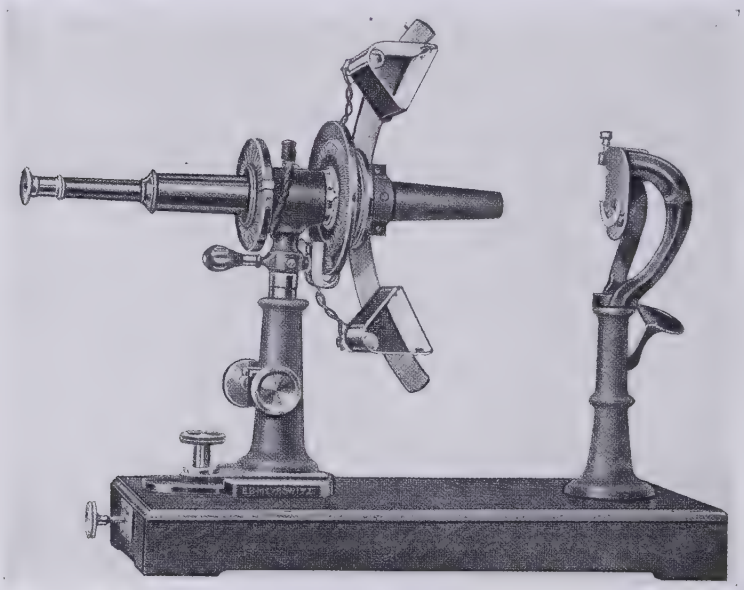


Fig. 427.—Javal-Schiötz ophthalmometer (model of 1907).

gives a clue to the axes of the meridians of least and greatest corneal curvature, as well as the amount of astigmatism.

Not infrequently the instrument indicates that the principal meridians of the cornea are not at right angles to each other—for example, it may record + 3.00 D cyl., axis 80°, or —3.00 cyl., axis 180°. In these cases, when there is hyperopia, the axis of the cylinder should be 80°, and when there is myopia, 180°.

In patients with heavy overhanging lids, deep-set eyes, or long lashes it is at times extremely difficult or even impossible to measure the vertical meridian of the cornea with the ophthalmometer.

Nothing is more common than to see the mires separate and overlap again, so that the apparent curvature of the cornea seems to change while under observation. This change is due to slight movements of the eye which bring different portions of the cornea into view. It is difficult for most patients to remain long in the required position before the instrument, and hence the readings should be rapid as well as accurate.

As to the correspondence between the amount of corneal astigmatism indicated by the ophthalmometer and the total astigmatism under a mydriatic, there is a difference of opinion among observers. Probably the rule formulated by Burnett is, in the main, correct: "For the total subjective astigmatism, subtract 0.50 D from the corneal astigmatism when it is according to the rule, and add 0.50 D if the corneal astigmatism is against the rule."

In addition to the Javal-Schiötz ophthalmometer a number of excellent models may be obtained. To some of these brief reference has been made on page 120. In so far as the author's practice is concerned his best results have been obtained with the Javal instrument (Fig. 427).

The ophthalmometer is exceedingly useful, and one of the most important of all the instruments of precision we possess for the diagnosis

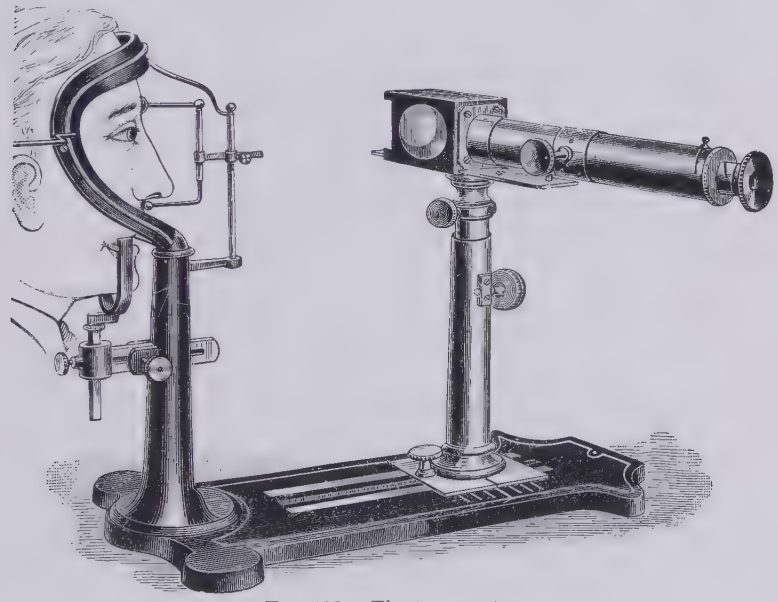


FIG. 428.—The tropometer.

of astigmatism of the cornea; but it should never be used for the prescription of glasses to the exclusion of other methods—the trial-lenses after mydriasis and retinoscopy.[1]

The Use of the Tropometer.—Dr. G. T. Stevens[2] attached special importance to the determinations, absolute as well as comparative, of the rotations of the eyes, since he believed that excessive tensions upon the vertically acting muscles of the eyes often induce converging or diverging strabismus, independently of any anomalous

[1] For a thorough exposition of the principles of keratometry the student should consult Carl Weiland, Archives of Ophthalmology, vol. xxii, pp. 37–64; Optique Physiologique, by Tscherning, pp. 46–68.

[2] International Ophthalmological Congress, Edinburgh, August, 1894; Annales d'Oculistique, April and June, 1895.

tension of the laterally acting muscles, and that many conditions of heterophoria may be explained in a like manner.

The most favorable rotations, according to the late Dr. Stevens, are: upward, 33°; downward, 50°; inward, 55°; outward, 50° (compare with page 603).

He devised an instrument, called the tropometer (Fig. 428), for the determination of the various rotations. The description of the method of using this instrument was kindly revised by him.

The instrument consists essentially of a telescope in which an inverted image of the eye is found at the eye-piece, where its movements can be observed upon a

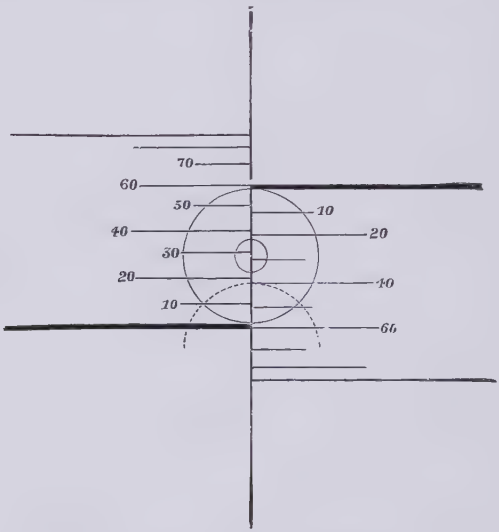


FIG. 429.—The long line between and at right angles to the shorter lines divides two similarly graduated scales running in different directions. The larger circle represents the outer border of the cornea, the edges of which are in contact with the two strong lines. The interval between each pair of short lines of the scale is ten degrees of an arc, commencing at the strong line in each case. If, now, the head of the person examined is held firmly in the primary position, and the eye caused to rotate strongly in a given direction, the arc through which the border of the cornea passes may be accurately read upon the scale. In the figure the curved dotted line represents a new position of the border of the cornea. Suppose that the person examined has been directed to look strongly upward. Then the cornea has moved *down* the scale, and reaches the point in this example of 40°, that being the measure of this rotation.

By means of the small lever the scale can be placed horizontally, vertically, or obliquely, and by means of the two graduations measurements in opposite directions can be made.

If it is desired to determine the upward rotation, the border of the cornea is made to coincide with the strong line which appears in the upper part of the scale at the right hand. This adjustment is made by means of the milled head at the side of the standard. As the eye rotates up, the image moves apparently down. In determining the downward rotation the strong line at the lower left-hand side of the scale is taken as the point of departure. For lateral rotations the scale is turned to the horizontal position, and the corresponding strong lines used as before.

graduated scale, permitting rotations in any direction to be measured. A prism or a diagonal mirror at the objective end of the telescope permits the observer to sit at the side of the observed. By means of a head-rest and an adjustable stirrup with a

wooden bar, which the observed holds closely between the teeth, the head may be held firmly in the primary position. This position is indicated by the two buttons at the extremities of the guiding rods.

In order to adjust the upper border of the cornea to the line, it will generally be necessary for the examiner to place the left hand upon the forehead of the patient and make gentle traction of the upper eyelid by the thumb. An application of the hand to the head is advisable in all measurements, as by this means the examiner is able to detect even a slight movement of the head, which would vitiate any measurement of the rotation.

In adjusting the head to the head-rest the teeth should be closed upon the wooden bar of the stirrup with force; then, after adjusting the stirrup to the proper height, the two indicators should be adjusted, one touching the glabella or ridge just above the root of the nose, the other pressing the commissure of the upper lip close below the nose. By pushing the stirrup forward or backward the lower indicatory button should be at a distance from the bone equal to that of the upper indicator.

The hoop passing around the head is designed to indicate, when the knob presses against the occipital protuberance, that the head is in position for lateral measurements.

If the cornea is large, the telescope must be moved backward upon the base until the borders of the cornea just encroach upon the two strong lines of the scale. When the cornea is small the tube is moved forward.

The wooden bar of the stirrup may be thrown away after use and replaced by another.

For measuring declinations of the retinal meridians Dr. Stevens has designed an instrument known as the *clinoscope*. For a full description of this instrument and the manner of using it the student should consult the Medical Record, February 16, 1901, where he will find Dr. Stevens' complete directions.

Localization of Foreign Bodies in the Eyeball with the Roentgen Rays.—The following paragraphs have been written by Dr. William M. Sweet, and, therefore, in his own words describe the method which he has originated and which has proved to be most satisfactory:

The methods of locating foreign bodies in the eyeball by means of the Roentgen rays are based upon the study of the shadow of the foreign substance on the radiograph in its relation to the shadow of one or more known points in the vicinity of the eyeball. These fixed points from which measurements are made may be situated on the skin of the eyelid or cheek, or suspended in front of the eyeball. If an apparatus is employed to fix the position of the *x*-ray tube at each exposure, only one indicating point will be required, but with two fixed points of measurement the position of the tube at the time the radiographs are made need not be known.

The earlier form of the Sweet localizing apparatus consisted of a small platform to which the head of the patient was firmly clamped. Two ball-pointed indicator rods were used, one opposite the center of the cornea, and the other at a fixed distance to the temporal side. Two exposures were made, and the situation of the steel in the eye was determined from a study of the position of the shadow of the foreign body on the two plates in relation to the shadow of the two indicating rods.

A new form of apparatus has been designed in which only one indicating rod is used. The planes of shadow of the foreign body are accurately determined by the instrument without the necessity on the part

of the operator of taking measurements from the plates or in drawing lines on the chart. The tube-holder, indicating ball, and plate-holder are upon a movable stage, and therefore preserve a known relation to each other which does not vary. The angle of the rays with the eye-ball and the distance of the tube from the plate are always the same, so that one indicator is sufficient, and this consists of a small steel ball supported in a ring of transparent celluloid. The setting of this ball opposite the center of the cornea is made by means of adjusting screws conveniently placed on the frame of the instrument. Accuracy in the measurement of the distance of the indicating ball from the center of the cornea is secured by means of a telescope and reflecting mirror. The mirror gives an image of a cross-wire and a lateral image of the cornea. Through the telescope the observer adjusts the instrument until the image of the cross-wire is in direct contact with the image of the summit of the cornea (Fig. 430). When the adjustment is made, the indicating ball is exactly 10 mm. from the center of the cornea. A miniature incandescent lamp, mounted in an adjustable shade, illuminates the side of the nose of the patient, insuring a well-lighted image of the cornea and cross-wire.



Fig. 430.—Image of cross-wire and cornea (W. M. Sweet).

Instead of a ball of cotton or other object for fixation, as in the older method, a circular mirror is placed at a distance of 12 inches above the injured eye. The patient gazes in the mirror and sees a reflected image of the injured eye and the circular celluloid disk with the steel indicating ball in its center. After the ball has been adjusted to a point opposite the center of the cornea of the injured eye, the patient by fixing the ball with the seeing eye prevents any movement of the eye during the exposures and holds the visual line of the injured eye parallel with the plate. Two exposures are made upon one plate, metallic shutters protecting those portions of the plate which are not to be exposed to the rays.

The tube-holder contains the usual cylindric lead-glass shield for protecting the operator from the action of the rays, with the customary lead diaphragm. The central orifice of the diaphragm is covered with aluminum, which offers little obstruction to the rays, but lessens the risk of any unfavorable action of the rays upon the patient and guards against possible damage to the eyes in the event of breakage of the tube. The tube-holder slides upon a graduated rod, and the first exposure is made with the indicator at zero, in which position the rays pass in a direction corresponding with the horizontal plane of the eye-ball. The second exposure is made with the tube at its farthest point

to the right or left of the first position, depending upon which eye is to be examined.

Since the relative position of the tube in reference to the indicating ball and the photographic plate remains fixed and known, the x-rays in passing through the eyeball follow a definite course, which is always the same for the two separate exposures. It is, therefore, possible to indicate on the localization chart the direction of the rays at the two exposures, and this has been done in the chart, a copy of which is repro-

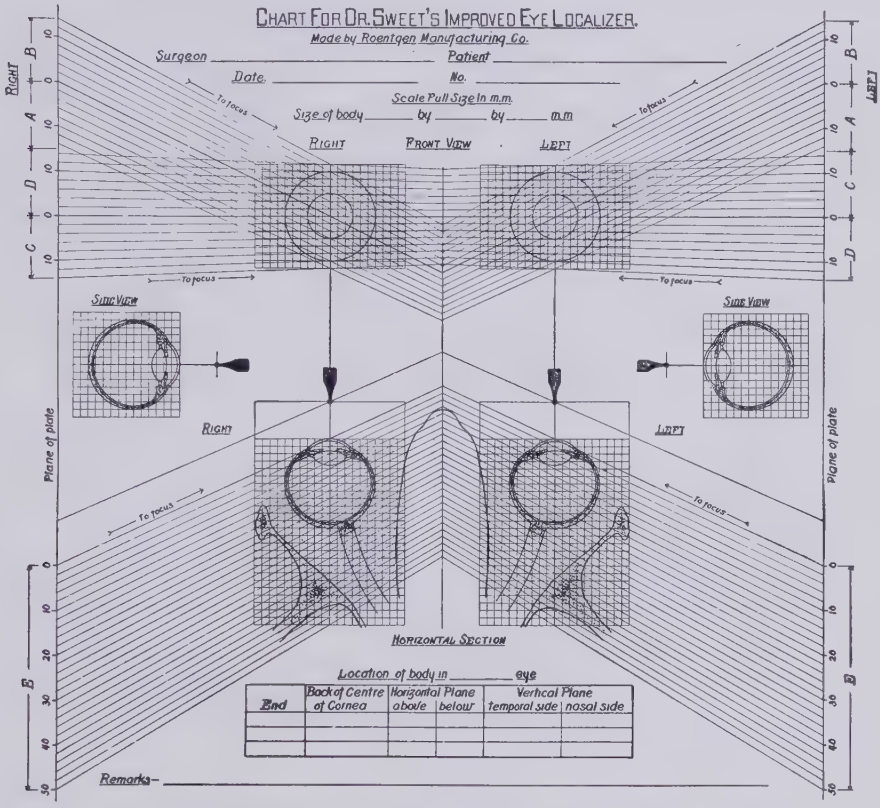


FIG. 431.—Localization chart, with lines representing course of the x-rays (one-half actual size) (W. M. Sweet).

duced in Fig. 431 reduced in size one-half. Only those lines representing rays 2 mm. apart are reproduced, but each line is drawn with the required amount of divergence to indicate the rays as coming from a point the distance of the tube from the photographic plate.

In employing the new apparatus (Fig. 432) the jointed portion containing the upright tube, T, is raised, and the patient lies with the head on a platform of hard fiber, with a pillow beneath the shoulders and a small sand-bag under the head and neck. The upright supports for holding the head are now adjusted by means of the wheel 1, and the

jointed part of the apparatus brought down in position. The indicating ball is now roughly adjusted until it is opposite the center of the cornea and about 12 or 15 mm. distant. The patient looks with the uninjured eye into the mirror (*M*) and fixes upon the iris or cornea of the injured eye, or, better, upon the indicating ball in the center of the celluloid disk. The indicating ball is now adjusted directly over the corneal center by means of the wheels 2 and 3, and the correctness of the position verified by observation through an opening in the mirror (*M*). The operator then adjusts the light of the small electric lamp so that the side of the nose next the injured eye is illuminated, but the light is not thrown into the eye. With this area lighted it is possible,

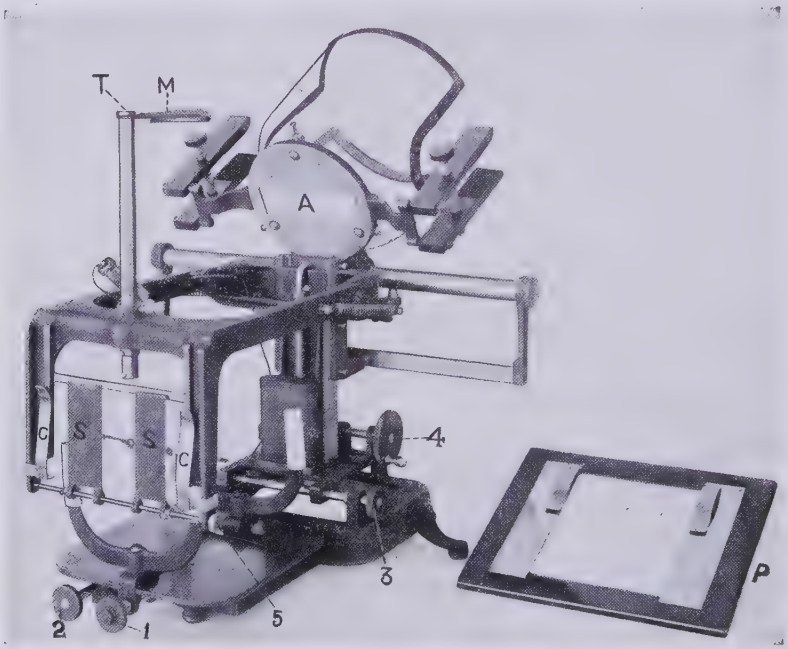


FIG. 432.—Sweet's apparatus for localizing foreign bodies in the eyeball.

through the telescope (*T*), to note when the cross-wire is exactly tangent with the summit of the cornea. The movement necessary to secure this position of the wire is made by means of the adjusting wheel 4. When the image of the cross-wire touches the image of the corneal summit, the indicating ball is exactly 10 mm. from the eyeball.

The photographic plate is inserted beneath the spring clips (*C*, *C*), the shutters (*S*, *S*) moved so that the center area is open, and the tube-holder adjusted to the zero point on the sliding scale. The current is turned on, and one exposure made. The tube-carriage is then moved to the limit of the sliding rod, always in the direction of the chin of the recumbent patient (to the end marked *R* if the radiographs are made of the right eye, and to *L* if of the left eye). The upper shutter is moved

to cover the exposed central portion of the plate and uncover the upper unexposed portion. The current is again turned on and the second exposure made.

After the plate is developed it is placed in the frame *P* (Fig. 432), containing the key plate or focal co-ordinates (Fig. 433), with the film side of the radiograph next to the key plate. The radiograph is moved until the shadow of the indicating ball of the first exposure is in apposition with the middle ball on the key plate and the heavy horizontal line of the radiograph parallel with the horizontal line on the plate. Holding the frame to the light, there is noted the position occupied by the shadow of the foreign body with respect to the vertical lines of *C* and *D*. A reading is made of the line or lines which pass through

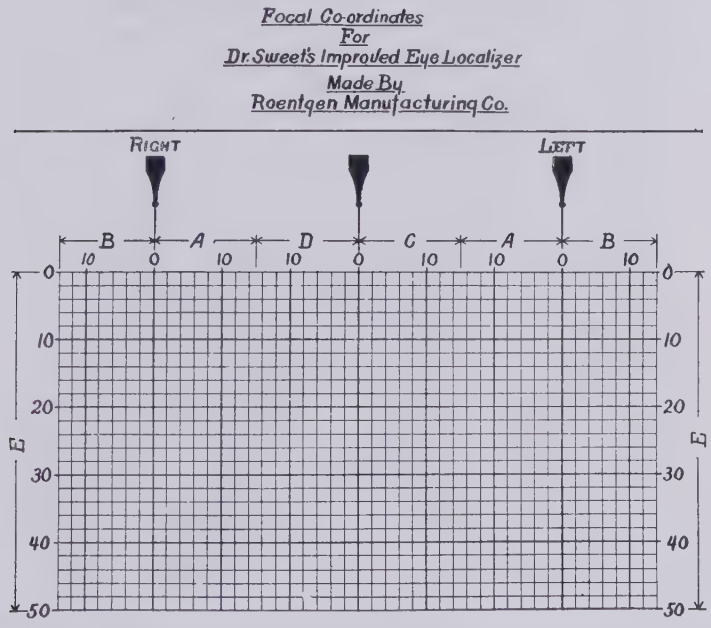


FIG. 433.—Plate showing focal co-ordinates (three-fourths actual size) (W. M. Sweet).

the body, and this is transferred to the corresponding lines of the *C* or *D* scale of the chart, to the right or left side, depending on which eye is under examination. Without moving the plate the *E* reading is similarly made and transferred to the chart. To take the *A* or *B* reading the plate is shifted slightly until the image of the indicating ball on the second exposure coincides with the "Right" or "Left" ball of the vertical co-ordinates *A* or *B*. The line or lines of the *A* or *B* co-ordinates which cross the shadow of the body are noted and indicated on the *A* or *B* lines of the chart. The horizontal co-ordinate *E* should be the same in both readings. If the focus point on the anode of the tube has been accurately set by the cross-lines on the lead-glass shield of the tube-holder, the images of the indicating ball on the plat

will coincide simultaneously with those on the transparent key plate, and it will then not be necessary to reset the plate to read the position of the *A* and *B* co-ordinates.

After the three readings have been transferred to the chart, the point of crossing of the *A* or *B* and the *C* or *D* lines is found, which gives the location of the foreign body in reference to the front view of the eyeball, indicating its situation above or below the center of the cornea and to the nasal or temporal side of the vertical plane. Where a vertical line from this point crosses the *E* reading on the horizontal section of the globe it gives the depth of the body in the eyeball or

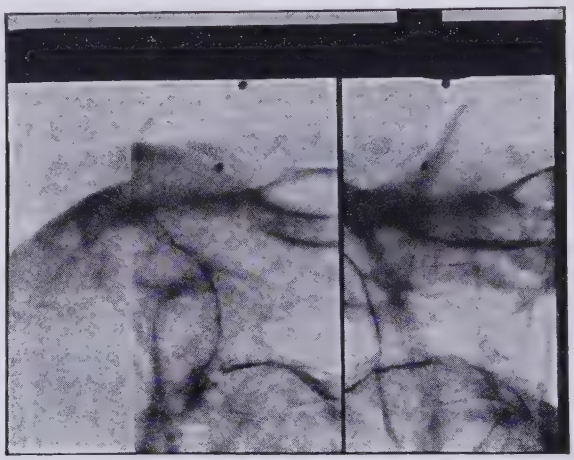


Fig. 434.—Radiograph of foreign body in eye (three-fourths actual size) (W. M. Sweet).

orbit. In bodies of large size both ends should be localized to give the position in which the body rests in the globe. The situation of the body on the side view is determined by transferring its measured depth from the horizontal section and its distance above or below the horizontal plane from the front view localization.

The accuracy of the localization depends only upon the care with which the operator adjusts the indicating ball opposite the center of the cornea and at the definite and fixed distance from it. After the exposures are made and the plate developed, the determination of the situation of the foreign body is simply a question of reading from a key plate and transcribing these readings to the chart.

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